THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—31ST YEAR. SYDNEY, SATURDAY, JANUARY 29, 1944. No. 5.

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REMARKS CONCERNING VITAMIN DEFICIENCY IN THE AUSTRALIAN ADULT.[1]

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To review in twenty minutes the vast and rapidly mounting literature concerned with deficiency disease is a task far beyond my capacity. I shall therefore limit my remarks to some generalizations of a broad character and to a few observations of topical interest and importance.

Deficiency disease may arise from deprivation of basic food constituents, such as water, protein or carbohydrate, minerals such as sodium, calcium and iron, hormones either endocrinal or alimentary, and vitamins. Tonight I propose to refer to vitamin deficiency alone.

While much of this subject is still controversial and in a state of flux, there is no other branch of medicine which has progressed further in the last ten years than that of nutrition. Wartime feeding problems have accelerated rather than retarded this development. There has been a world-wide awakening to the fundamental importance of adequate nutrition, and this must eventually lead to the establishment of a national and individual nutritional conscience, which may go far towards the eradication of deficiency disease. The magnitude of the malnutrition problem has been established in three ways: (i) by food surveys, (ii) by hospital and mortality statistics relating to deficiency disease, (iii) by laboratory investigations upon subjects fed collectively.

In food surveys, a sample of the community is invited to keep a food diary, and the adequacy or otherwise of the food bought is assessed quantitatively. Such surveys can at best lead to only the broadest conclusions, because of meals eaten away from the house, wastage in cooking and unconsumed food.

Hospital and mortality statistics relating to deficiency disease must considerably under-estimate the incidence of these disorders because many such diseases are listed under different headings—for example, toxic polyneuritis, psychosis, secondary anæmia *et cetera*. The problem is one of medical reeducation.

Laboratory investigations upon subjects fed collectively are by far the most accurate method; but obviously they cannot apply to the average citizen, who has a free choice in the open food market. Similar surveys are needed of such subjects selected at random. A model survey of this type was recently completed in a Royal Navy mine sweeper, in which all the crew received food from the same mess. The weight of each amount of food on each man's plate was recorded, and also the unconsumed items of food after each meal. No food was eaten away from the ship. Samples of the food as eaten were submitted to vitamin analysis at a shore laboratory, checks were made on stores consumed and observations were conducted on the mode of cooking. At the end of the trial period vitamin analyses of the blood and other laboratory tests were carried out.

Evolution of the Concept of Vitamin Deficiency.

The acceptance of a human malady as due to a single or multiple vitamin deficiency has been in part attributable to animal experimentation with deficient diets, in which syndromes comparable to those observed in certain types of human illness have made their appearance and have been cured by subsequent addition of the missing food factor (for example, beriberi, rickets). Some animal syndromes have not yet reflected a known human symptom complex (for example, chick dermatitis); in other instances the same vitamin deficiency is responsible for quite different syndromes in the animal and in man (for example, black tongue in dogs and pellagra in man). Less striking but even more fundamental effects on growth and resistance to disease are common to many experimental vitamin deficiencies, and may prove to be of equal importance to the human organism. Commercial distributors of vitamin preparations would have us believe that nearly all the morbid signs and symptoms shown by the deficient animal have their literal counterpart in man, and that dosages for human consumption can be accurately assessed from the results of such experiments. We must be cautious, however, in the acceptance of such conclusions.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 26, 1943.

I suggest that unless a "deficiency disease" can fulfil the following postulates, it cannot be accepted as a specific clinical expression of mono-vitamin shortage:

1. The syndrome must be faithfully reproduced in all subjects by complete withdrawal of the vitamin or vitamin complex, after an approximately equivalent minimum of time, and irrespective of age, race, environment or associated disease.

2. The subject must be completely restored to the original state of health by adequate absorption, in a pure state, of the vitamin omitted, providing a late irreversible stage has not been reached.

3. The symptom complex so produced must not be wholly curable by any other agent than the specific vitamin concerned.

4. The deficiency substance must be found in the natural food or water intake of the subject, and the deficiency state must be equally well cured by a sufficient absorption of such food.

There may be other restrictions; but, applying these alone, we limit the list of proven deficiencies to florid deficiencies—for example, xerophthalmos and degeneration of other ectodermal tissues (vitamin *A*), degeneration of nervous and cardiac tissues (vitamin B_1), degeneration of the mucosa of the alimentary canal (vitamin B_2, B_6 *et cetera*), degeneration of matrix-forming tissue (vitamin *C*), degeneration of cartilage (vitamin *D*), degeneration of clotting factors (vitamin *K*). These degenerations ultimately assume a constant clinical type, even in the presence of associated disease of another character. The proof of their identity does not rest upon animal experiment, induction or hypothesis, as still remains the case with deficiencies in certain other vitamins and filtrates—for example, pantothenic acid, biotin, pyridoxine *et cetera*. They can be cured by synthetic as well as by natural vitamins. The commercial interests with new vitamins to sell for old have compiled extensive lists of psychiatric, neurological and dermatological factors, many of which cannot fulfil even one of the postulates described. There is thus a danger that vitamin therapy, which today has been so much simplified, may be so loosely applied as to be blinding, wasteful, expensive, even dangerous.

The Clinician's Contribution.

It must not be forgotten that long before nutritionists began experimenting on animals with deficient diets, shrewd clinical observation and trial had gone far to ascertain the nature and means of relief of scurvy and endemic pellagra. Since research chemists have made available synthetic vitamins of high potency, clinicians have been able to dissect beriberi, pellagra and sprue into their component deficiency syndromes. Animal experimentation and laboratory diagnosis have made contributions of great magnitude towards the recognition and treatment of deficiency disease; but clinical acumen and judgement have been, and always will be, the hinge upon which the door swings to exclude this form of illness.

Recognition and Diagnosis of Vitamin Deficiency.

Logical anticipation of the possibility that a deficiency syndrome may be present is of the highest clinical value. As in the diagnosis of hyperthyreoidism, myxœdema, bacterial endocarditis and general paralysis, recognition will only come swiftly to the prepared mind. It is wise to make a rule that the diagnosis of a vitamin deficiency should not be made on the basis of a single sign, but that careful search should be made for corroborative clinical evidence. Diet histories are only a rough guide, but are obviously indispensable. An apparently satisfactory dietary forms no guarantee that vitamin deficiency can be discounted. We must even bear in mind that an accurate food history is exceptional, that the same food from different sources and seasons may vary widely in its vitamin content, that handling, storage and methods of cooking can achieve a high reduction in vitamin content. When we add the possibility of difficult absorption and even of increased requirements or of destruction after absorption, we can realize that vitamin deficiency may subtly lift its head in most unexpected surroundings. National and individual economics, food faddism, diarrhœa, peptic ulcer and other alimentary disorders, liver disease or biliary obstruction, mental affliction, alcoholism, surgical interference with intestinal absorption, all can be expected to contribute instances of vitamin deficiency. Subjects such as diabetics, the obese, dyspeptics, and others taking fixed diets, are to be suspect, as such diets are often unscientifically prepared and are pursued by conscientious patients for periods much longer than intended by the prescriber. Chronic toxæmias and long debilitating illnesses, which depress appetite and intestinal function, may bring about nutritional defects. Anorexia in acute diseases never lasts sufficiently long to produce signs of vitamin inadequacy, but as vitamin needs are increased, it may be enough to precipitate an acute deficiency syndrome if vitamin stores are low. Prolonged starvation, partial or complete—for example, in *anorexia nervosa*, in isolated military units with a bad supply service, in the case of a few settlers in far outback and in that of shipwrecked mariners *et cetera*—is prone to bring about evidences of vitamin *B* or *C* deficiency. The tendency of the senile to eat monotonously similar meals from the one kitchen, combined with their decrescent absorptive faculties, exposes them to this type of disorder. Relative insufficiency may be anticipated during periods of increased vitamin need—for example, during pregnancy and lactation.

The general practitioner is in the best position, with his knowledge of the patient's economic situation and food habits, to suspect an early deficiency disease and so to achieve a therapeutic triumph; but according to the site and character of the earliest symptoms, patients tend to present themselves to certain specialists. In the case of vitamin deficiency, the dermatologist, psychiatrist, ophthalmologist, orthopædic surgeon and gynæcologist are some of the consultants who may occasionally encounter virgin cases of deficiency disease. If they wish to avoid missing an unusual therapeutic opportunity, they must inform themselves carefully of the clinical character of vitamin deficiencies encountered in their own field. Thus the eye specialist will see early limbal vascularization in riboflavin deficiency, the xerophthalmia of vitamin *A* lack, and the cataract accompanying disturbances of calcium metabolism; the dermatologist may be consulted about the pigmented hyperkeratosis of knees or elbows or about the perioral fissuring and nasolabial sebaceous plugs found in vitamin *B* subnutrition; the gynæcologist may be consulted about epithelial metaplasia in and around the female genital tract; while the radiologist may observe "deficiency patterns" of the intestine, bony decalcification and deformities, or signs of active or healed lesions about the epiphyses. The psychiatrist and the neurologist must be prepared to recognize the "neurasthenic" syndromes so frequently encountered in early deficiency states, as well as the psychosis and peripheral nerve lesions of later phases of hypovitaminosis *B*. Orthopædic surgeons require to become familiar themselves with the details of an adequate diet if they wish their mechanical successes to be permanent. Rhinologists have to recognize the snuffling and coughing of semi-rhachitic toddlers.

In man we must not expect to recognize the pure syndromes described as athiaminosis, ariboflavinosis *et cetera*, encountered after deliberate exclusion of one single vitamin from the diet. Foods and their relationship, before and after consumption, are highly complex. A lack of one ingredient may interfere with the use of others which are present; for example, the advent of rickets is facilitated by a high carbohydrate intake. The amount of thiamin and probably niacin and riboflavine absorbed must be proportional to the caloric and carbohydrate intake. While it is of fundamental importance to search for all the units of a clinical syndrome, as described in a text-book, we must not expect to find more than one or two. Confirmation of the presence of a deficiency can in some cases be safely made by the laboratory or X-ray studio (see Table I); this applies, for example, to vitamins B_1, *C* and *D* and *K*. In other cases tests so far employed have been complicated and unsatisfactory (for example, nicotinic acid and riboflavin deficiencies). In the absence of such facilities, the thera-

TABLE I.
Specific Vitamin Deficiencies and Their Detection.

Vitamin.	Chief Source.	Chief Deficiency Symptom.	Laboratory Test.[1]	Common Associated Deficiencies.	Relation to 100 Calories (Intake).
A	Carotene from yellow legumes, butter and dairy products, cod-liver oil, halibut liver oil.	Poor dark adaptation. Xerophthalmia. Night blindness. Phrynodermia.	Vitamin *A* tolerance test. Biophotometer.	Vitamin *D*.	
B complex. B_1 (thiamin)	Pork. Milk. Yeast.	Beriberi. Peripheral and central nerve lesions.	(i) "Tolerance test" after 5 milligrammes of thiamin.	Usually all vitamin *B* factors about same nutritional status.	50 to 70 microgrammes.
B complex. B_2 (riboflavine)	Liver. Wheat germ.	Pellagra. Cheilosis.	(ii) 24-hour excretion, after same dose.		500 to 800 microgrammes.
B complex. B_3 (niacin), pyridoxin, choline, pantothenic acid	Rice polishings *et cetera*.	Corneal vascularization.	"Tolerance test" after 150 to 300 milligrammes of niacin.		70 to 100 microgrammes.
C	Germinated peas, citrus fruit, cress, parsley, pawpaw, black currants *et cetera*.	Scurvy. Slow healing of wounds.	24-hour excretion or factor thereof.	Vitamin *P*.	
D_3	Sunlight, cod-liver oil, irradiated dehydrated cholesterol.	Rickets, osteomalacia.	(i) X-ray study. (ii) Serum phosphorus, and calcium depressed. Serum phosphatase elevated.	Vitamin *A*. Vitamin *B*.	
E	Wheat germ.	? Habitual abortion.			
P (hesperidin)	Lemons; orange peel.	Purpura.	Capillary fragility tests.	Vitamin *C*.	
H (biotin)					
K	Leafy vegetables.	Hæmorrhage following operation for liver disease. Hæmorrhagic disease of newborn.	Prothrombin time.	Vitamins *B*, *C*.	

[1] Some of these laboratory tests, for example for ariboflavinosis, vitamin *A* deficiency *et cetera*, are not yet perfected.

peutic test, preferably made without the patient's knowledge, is generally the easiest and best. Specific therapeutic substances of high concentration are employed, preferably parenterally. If real vitamin deficiency is the cause of the illness, rapid improvement should occur by after ten to fourteen days, unless the disease has reached an advanced and refractory stage, when heavy intravenous doses of vitamins may become necessary. Certain conditions formerly recognized or suggested as examples of deficiency disease are no longer to be found in this category.

From a review of recent literature, it appears that night blindness is not now recognized as due entirely to vitamin *A* deficiency, and that capillary fragility appears to be an expression of vitamin *P* deficiency rather than of vitamin *C* deficiency. Vitamin *E* lack is not the cause of progressive muscular atrophy. A good case has been made out for the acceptance of sprue, idiopathic steatorrhœa and cœliac diseases as a common expression of nicotinic acid deficiency. A magenta-coloured tongue is now said to be characteristic of riboflavin shortage. Cheilosis and dermatitis of the lips are common to niacin, riboflavin and pyridoxin insufficiency, while Vincent's angina is held to be grafted on the ulcers of a stomatitis due to nicotinic acid lack. Wernicke's encephalopathy, with a fatal outcome encountered in advanced alcoholics and psychotics, and characterized by progressive stupor, cogwheel rigidity of the extremities, grasping and sucking reflexes, is considered to be a terminal form of deficiency disease, chiefly lack of nicotinic acid. Vitamin *A* should no longer be called the anti-infective vitamin, as this property is common to others (for example, vitamins *B*, *C* and *D*) and any relation between vitamin *A* deficiency and urinary lithiasis has been abandoned. Pregnancy toxæmia does not appear to be associated with vitamin shortage, while in some quarters it is held that hepatic cirrhosis may be a deficiency disease. Doubt as to the ability of lack of thiamin to produce complete polyneuritis has crept into recent literature.

Latent or Partial Vitamin Deficiency.

Many of the gross forms of deficiency disease develop suddenly as an acute illness and with little previous warning of any serious departure from health. On the other hand, laboratory, X-ray and vitamin excretion studies can early reveal a paucity of circulating and stored vitamin. Considerable controversy has arisen as to the clinical significance of these findings. It is obvious that in the case of certain vitamins—for example, vitamins B_2, *C* and *D*—there is a wide safety margin between the so-called "biochemical lesion" and frank clinical signs. The point at issue is whether growth and resistance are better at levels of vitamin intake which will ensure saturation, or whether a considerably lower consumption is adequate—or, more briefly, whether biochemical or clinical standards are to prevail. In my own view this question cannot be answered in a simple form. Some vitamins—for example, B_2, B_3 and *D*—are stored in the tissues; others, such as vitamins B_1 and *C*, are excreted rapidly. Further, there are certain individual and perhaps racial idiosyncracies and adaptabilities to be considered. Vitamin *C* shortage is tolerated much better than vitamin B_1 shortage. It may be that nervous tissue is more sensitive to the shortage of enzymes concerned in its metabolism than less highly differentiated mesodermal tissue. Great numbers of nutritional surveys conducted in Europe and the United States of America have revealed suboptimal vitamin intake. Reports of nutritional studies repeated since the outbreak of war have shown progressive deterioration in vitamin stores in groups of school children, naval ratings and civilian adults, without any apparent diminution in health or capacity for effort. None the less, studies of this character made in small groups of individuals do not necessarily reflect the nutritional status of the community as a whole. Mortality statistics, especially for pulmonary tuberculosis, are rising sharply in England, and it is surely more logical and safer to make every effort to correct any possible contribution towards such a situation made by subnutrition. This effort is being made in the United Kingdom and also, we are told, in Germany. The national loaf, the "victory garden" and scientifically planned food rationing has kept wartime deficiency disease, in England at least, to a highly commendable minimum, although there has undoubtedly been some depression in the general health level of the nation. The German method has been the far easier but less satisfactory universal issue of synthetic vitamin tablets. Correction of such subclinical deficiencies must constitute one of the foundation stones of enlightened post-war nutritional planning.

Deficiency Disease in Australia.

Gross deficiency disease is probably less common in Australia than in almost any other country in the world. We have all encountered isolated examples of scurvy,

nutritional macrocytic anæmia, non-tropical sprue *et cetera*; but florid vitamin deficiencies at the stage of functional and structural damage are in my experience uncommon in this country, and certainly in this State. I have searched unsuccessfully for some years for a case of "beriberi heart". In normal times there should be a sufficiency of protective foods for all social levels, and while individual preference, misdirected thrift and great isolation may produce partial deficiencies in respect particularly to vitamins *C* and *D*, we are not faced with problems such as the endemic florid deficiency disease which occurs in the southern United States of America, the Far East, Russia *et cetera*. Thus the reports of the Royal Prince Alfred Hospital during the last thirty years show only ten cases of scurvy, twenty of beriberi (all in Chinese) and two of pellagra. Sporadic instances of advanced rickets and hæmorrhagic disease of the newborn have occurred more frequently in specialized hospitals. Country practitioners in general are probably more likely to encounter florid deficiency disease, especially those in the outback; but as large eaters of meat and potatoes, we have a good start on other nations. On the contrary, I am convinced that in both service and civilian practice, partial vitamin deficiencies are not recognized in more than half the patients affected. Some of these deficiencies are mainly "biochemical lesions", such as the vitamin *C* subnutrition which has proved to be common in sea-going personnel of both the Royal Navy and the Royal Australian Navy, and in many civilian communities in Great Britain. Such a long latent period exists before the first actual signs of scurvy appear, that this form of deficiency, as demonstrated by blood plasma and urinary saturation tests, appears to be of small practical significance. Even its relationship to gingivitis and chronic skin infections is doubtful. No longer can a modern Camoëns[1] complain in the following terms:

> Never mine eyes such dreary sight beheld,
> Ghastly the mouths and gums enormous swelled,
> And instant, putrid like a dead man's wound,
> Poisoned with fœtid streams the airs around,
> No sage physician's ever watchful zeal,
> No skilful surgeon's gentle hand to heal
> Were found: each dreary mournful hour we gave
> Some brave companion to a foreign grave.

Of greater significance are the early signs of vitamin B_1 deficiency, which are appearing particularly in troops stationed in tropical areas. The symptoms may contribute to the state of general lassitude, loss of weight, incapacity for sustained efforts, depression and irritability common to personnel quartered for a year or more in the northern bases and combat areas—for example, the "Darwinitis" of 1940-1941 and perhaps *le cafard* of North Africa. More severe forms include the nutritional œdemas, peripheral nerve palsies, and sensory disturbances sometimes unassociated with malaria which I have seen occasionally in officers and men of the Royal Australian Navy. Bread for such areas requires to be enriched with vitamin B_1, or additional wheat germ must be provided. There are two other forms of vitamin *B* subnutrition encountered in civilian practice which repay diet investigation and energetic nutritional therapy. One is the smooth, glazed tongue, not necessarily associated with achlorhydria, diarrhœa, macrocytic or microcytic anæmia or dysphagia, which is seen in women of late middle age, and the other the recurrent buccal and labial ulceration affecting the same type of subject. Non-tropical sprue is occasionally encountered in this city and should respond favourably to the administration of nicotinic acid.

Without venturing far upon the ground of Dr. Greene's subject, I cannot refrain from referring to what I consider to be the commonest form of vitamin deficiency in New South Wales—namely, occult, but by no means latent, vitamin *D* subnutrition in infants, and to a less degree in pre-school children. While knock knee at the age of five years is not uncommon in the children of well-to-do parents, winter bronchitis, head sweating, muscular hypotonia, and delayed dentition and cranial ossification are frankly too common in all families, especially, however, in children of the public hospital class. "Sunny Sydney", partially screened by dust and smoke in the latter day winters, does not irradiate its overclothed children sufficiently to compensate for the inadequate unitage of vitamin *D* in its milk supply and in unstandardized cod liver oil.

Prophylaxis and Therapy.

It is now certain that gross deficiency diseases, such as beriberi, sprue, pellagra and scurvy, are all multiple deficiencies, and often will not respond completely to treatment with a single vitamin. Reference has already been made to the "therapeutic test" in the diagnosis of deficiency disease. Like all therapeutic tests, it should be applied only when clinical and ancillary tests have failed. Even if it appears to be successful, there are many possible fallacies. Thanks to the progress made in the synthesis of highly potent specific vitamins, we now have in our hands an amazing array of therapeutic armaments. These vitamins can be administered parenterally, and so uncertainties of absorption can be disposed of. They are of such concentration that, even if they are taken orally, relatively large amounts must gain entrance to the portal system. Water-soluble vitamins are rapidly excreted, and if diarrhœa is present, they may have to be given more often than once a day. Fat-soluble vitamins, if given by mouth, should be accompanied by bile salts to assist their absorption if liver or biliary insufficiency is suspected. The vitamins may be used singly or in combination according to clinical and biochemical indications. Proprietary vitamin mixtures vary so much in their composition, that it is essential to know their exact concentration, and often to fortify the content of the desired vitamin or vitamins. It is essential to think in terms of international units or milligrammes in respect to vitamin dosage, and not blindly to order drops or tablets. Quantitative rather than qualitative conceptions are called for today. A basic vitamin mixture which expresses the daily needs as commonly accepted is constituted as follows: thiamin, 10 milligrammes; niacin, 50 milligrammes; riboflavine, 5 milligrammes; ascorbic acid, 75 milligrammes. For beriberi 10 milligrammes of thiamin are added per day; for

[1] Camoëns, a Portugese poet, accompanied Vasco da Gama's first voyage to India about 1500.

TABLE II.

Numerical Data in Respect of Vitamins Required by Active Adult Male.

Vitamin.	Optimal Blood Level.	Daily Maintenance Dose.	Normal Daily Urinary Excretion.	Daily Therapeutic Dose.
A	60 to 190 international units per 100 cubic centimetres.	5,000 international units.	Negligible.	100,000 to 150,000 international units.
Thiamin (B_1)[1]	3·5 to 4·2 microgrammes per 100 cubic centimetres whole blood.	2 milligrammes.	80 to 300 microgrammes.	50 milligrammes.
Riboflavine (B_2)	35 to 45 microgrammes per 100 cubic centimetres whole blood.	3 milligrammes.	500 to 800 microgrammes.	10 milligrammes.
Nicotinic acid	10 to 18 microgrammes per cubic centimetre of red cells.	20 milligrammes.	3 to 10 milligrammes.	300 to 500 milligrammes.
Ascorbic acid (*C*)[1]	0·7 to 2·0 milligrammes per 100 cubic centimetres of plasma.	75 milligrammes.	15 to 50 milligrammes.	500 to 1,000 milligrammes.
K	(Prothrombin time, 90% to 100%).	——	Negligible.	5 milligrammes.
D	——	1,000 international units.	Negligible.	5,000 international units.

[1] One milligramme of thiamine = 333 international units; one milligramme of ascorbic acid = 20 international units.

scurvy, 100 milligrammes three times a day; and for mild pellagra, 50 milligrammes of niacin amide three times a day. A palatable, inexpensive "vitamin cocktail" can be prepared as follows, and it is acceptable even to acutely ill patients: two level teaspoonfuls of brewer's yeast powder, five drops of "Haliver" oil and "Viosterol", half a glass of citrus juice, sugar to taste; it is served three times a day. This cocktail, in addition to containing the "unknown" vitamins, provides the "known" vitamins as follows: vitamin B_1, 750 international units; vitamin B_2, 1,260 Sherman units; vitamin *A*, 25,500 international units; vitamin *D*, 5,100 international units; vitamin *C*, 1,320 international units.

Vitamin *A* deficiencies are best treated by halibut liver oil in doses of 10,000 to 20,000 units of vitamin *A*, while rickets is better corrected by ultra-violet rays or a solution of an activated dehydrocholesterol capable of providing at least 5,000 units daily. The danger of toxic overdosage from vitamin *D* has been over-estimated, and treatment by enormous single doses at longer intervals—100,000 units of vitamin *D*—has been equally successful.

Therapeusis by highly potent vitamin concentrates is often dramatically successful and rapid. There is little point in continuing an expensive method of treatment for longer than ten to fourteen days. Steps must then be taken to maintain an optimal vitamin level by dietetic means. Apart from the gustatory significance of properly prepared natural foods, the greatest value obtained from their consumption is that they supply the unknown factors along with the known. Further, we must distinguish between the mere use of natural foods and the proper combination of these foods, some of which are rich in one vitamin of the same complex and very poor in others. The same criticism applies to the prescription of vitamin concentrates, which give no greater security than the proper combination of natural foods. Minimal vitamin and mineral requirements are met by the provision of the following "protective" foods in the daily ration (McCollum): three-quarters of a pint of milk; four servings of fruit or vegetables, in addition to potato (one serving must be orange or tomato, one a green leafy vegetable, carrots or swedes); one egg, one serving of meat, fish or cheese (liver or kidney should be taken once a week); three teaspoons of butter, and whole grain cereal (for example, wholemeal bread, unrefined breakfast food *et cetera*). To go into further details as to methods of evaluating a diet, and the prescription of diets high in one or more vitamins for individual patients, is beyond the scope of this paper, and is actually the province of the trained dietitian, and of authors of text-books on nutrition. Only a few practical points can be given here. Full use should be made of wheat germ, dried brewer's yeast, unrefined liver extracts and commercial preparations such as "Marmite", "Vegex", "Bemax" and "Farex". Remember that serious responsibilities are the lot of any one undertaking the prescription of a special diet. If you are uncertain of the quantities required, do not specify any; or better, copy the diet in full from a reliable modern authority. All mothers should give their children 800 to 1,000 units of vitamin *D* per day. Bear in mind that butter is not a good source of vitamin *D*. Lean pork and dry legumes are the best sources of thiamin. Citrus fruits, pawpaw, cress, parsley, germinating peas and black currants are the best sources of vitamin *C*. Yeast and liver are the best and cheapest forms of vitamin *B* complex. Try to anticipate conditions under which vitamin deficiency is prone to occur and forestall its appearance by a thorough dietetic review.

Conclusions.

In conclusion, I trust that those discussing this paper will help to fill in its deficiencies. If I have introduced a new angle from which to view the chronic "neurotic" patient, I shall feel quite satisfied. At least I have studiously avoided biochemical formulæ, mathematics and the curious unpronounceable names of proprietary vitamins. I would suggest that consideration might be paid in the discussion to the question of the prevalence and present status of partial and fully developed deficiency diseases in this community, to the necessity or otherwise of radical changes in Australian food habits, to whether vitamins are being used intelligently and accurately in medical practice, to the necessity or otherwise for food fortification, and to experience of clinical data found to be of assistance in the recognition of hypovitaminosis.

Acknowledgement.

I am indebted to the Director of Naval Medical Services for permission to read this paper.

A CUTANEOUS REACTION TO THE INFLUENZA VIRUSES.[1]

By W. I. B. BEVERIDGE[2] AND F. M. BURNET,

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IN the course of work in this laboratory on intranasal immunization against influenza, occasionally symptoms have been observed which suggested that an allergic reaction was being produced. This led to our undertaking a series of experiments on the effects produced when influenza virus antigens in the form of specifically infected allantoic fluid or modifications thereof were injected intradermally in normal subjects. It was soon found that most adults gave an erythematous reaction, and that the power to produce this reaction was not abolished by boiling the allantoic fluid. A suitable reagent for most work was found to be a 1:10 dilution of allantoic fluid in saline solution. This was heated to boiling point for ten minutes, and amounts of 0·1 millilitre were injected, usually into the skin of the forearm. In a reactive subject an area of erythema 10 to 20 millimetres in diameter was produced, reaching its peak in twenty-four to thirty hours, after which it faded gradually. The erythema was frequently associated with slight swelling and sometimes with itching.

In view of the cutaneous reactions described by Frei for lymphogranuloma infection, and by Andervont and Rosenau[(1)] and by Wilson Smith[(2)] for vaccinia, it seemed probable that this reaction was essentially allergic in character and indicative of past infection with the virus. In order to determine whether the reaction was a specific response to influenza virus antigens, it was desirable first to show that it was not produced by material known not to contain influenza virus, and secondly, to show that purified or partially purified influenza virus retained the power to provoke the reaction.

The Specificity of the Reactions.

Tests with Non-Influenzal Control Material.

We have had considerable difficulty in obtaining fully comparable control material not containing influenza virus products. Normal allantoic fluid produced no reaction whatever in several adults who reacted to the influenza virus reagent. Such fluid, however, contains much less protein and none of the cell debris found in specifically infected fluids. On general grounds we considered that preparations made from the three antigenically distinct viruses influenza A, influenza B and Newcastle disease of fowls, all of which grow readily in the allantoic cavity, would provide mutual controls, since the physical character of the fluids was generally similar and the complete antigenic specificity shown in cross-neutralization or complement fixation tests would also be expected to manifest itself in allergic reactions.

We have tested a considerable number of specimens of human serum for antibodies against Newcastle disease virus, and except in the case of a laboratory worker known to have been infected (Burnet[(3)]), we have never detected

[1] Work carried out with the aid of grants for research on virus diseases from the National Health and Medical Research Council and from Mr. E. Alec Cato.

[2] Seconded from the Council for Scientific and Industrial Research, Division of Animal Health and Nutrition.

any trace of antibody. Nevertheless, when a Newcastle disease virus preparation was used as a control to influenza A and B intradermal tests, we found that 10% to 20% of adults gave reactions of about the same extent as those that followed the influenza virus injections. About half the remainder developed a slight degree of erythema, while the others did not react. It was noteworthy that hardly any of the children tested with Newcastle disease virus showed any erythematous response.

In an attempt to determine whether these anomalous reactions to Newcastle disease virus were due merely to a reactivity to normal chick embryo protein, many subjects were tested with several types of preparation from uninfected embryos. The most extensive tests were made with chorio-allantoic tissue ground up in saline solution, lightly centrifuged and boiled. After the boiling this contained 60 milligrammes of protein per 100 millilitres, about fifteen times as much as is present in the influenza virus antigen used. In other tests suitably diluted amniotic fluid (which has a high content of coagulable protein) was similarly used. In all, 53 tests with material containing from 20 to 80 milligrammes of normal chick protein per 100 millilitres were made. Only eight subjects showed any significant erythematous reaction, and in only two of these instances were the reactions comparable to those produced with influenza virus preparations. These two subjects (the writers) had received many experimental inoculations with chick embryo material of various types and had probably developed an abnormal reactivity.

The general impression obtained from these results was that, although a small proportion of adults undoubtedly show some erythematous response to injection of heated chick proteins, the reactions produced by the influenza virus preparations were so much more numerous and more extensive that they could not possibly be ascribed to normal embryo constituents. The significance of the Newcastle disease virus reactions cannot be usefully discussed until the detailed results of the tests have been described.

Inoculation of Partly Purified Virus.

Influenza B virus in the form of allantoic fluid with a hæmagglutinin (Hirst[6]) titre of 300 was passed through filter paper and centrifuged at 14,500 revolutions per minute for forty-five minutes. The deposit was resuspended in a small volume of saline solution and spun in the angle centrifuge at 3,000 revolutions per minute for fifteen minutes. The high speed supernatant fluid had a hæmagglutinin titre of 18; the final supernatant fluid after slow centrifugation had a titre of 320. When these fluids were diluted to 1:10, boiled and inoculated intradermally, both produced similar reactions of about the same size as the usual 1:10 reagent. Another batch of virus was passed through a gradocol membrane of an average pore diameter of 1·2μ and centrifuged at high speed as before, resuspended in saline solution and again centrifuged at high speed. The final deposit was suspended in a small volume of saline solution and had a hæmagglutinin titre of 320. A boiled 1:10 dilution of this fluid gave reactions the same size as those produced by the usual 1:10 reagent. The latter is prepared from fluids with a hæmagglutinin titre between 300 and 600.

Hirst, Rickard and Whitman[5] have reported that influenza virus adheres to the precipitate, largely composed of urates, which appears when infected allantoic fluid is stored in the cold. We took a sample of influenza A ("Melbourne") virus showing such a precipitate and centrifuged out the urates, which were washed once in a small volume of cold saline solution and again spun down, and then redissolved in warm saline solution. This fluid, which had a hæmagglutinin titre of 40, inoculated intradermally into three subjects, gave rise to reactions of the same size as the original allantoic fluid diluted to give an equal hæmagglutinin titre.

Another attempt was made to purify influenza virus B by adsorption in the cold to washed sterile fowl red cells with subsequent elution (Hirst[6]). Red cells were added to allantoic fluid to make a 3% suspension. This was well shaken and allowed to stand in melting ice for ten minutes; the cells were then washed three times with saline solution by spinning and resuspension, all the while being kept chilled. Finally the cells were resuspended in saline solution to one-third of the original volume, placed in the incubator at 36° C. and shaken every fifteen minutes. After three and a half hours the cells were spun down, a supernatant fluid with a hæmagglutinin titre of 160 being left. However, some hæmolysis of the cells had occurred, and when the fluid was boiled a coagulum formed. This was spun down and the nearly clear supernatant fluid was inoculated intradermally into four subjects together with a control made from boiling saline solution to which hæmolysed fowl cells had been added. In each person the control produced no reaction, but the virus-containing reagent produced a definite reaction.

Influenza B allantoic fluid virus diluted 1:3 was filtered through a Seitz E.K. pad. The filtrate, which gave no growth of virus when inoculated into eggs, and no hæmagglutination, produced no reaction on intradermal inoculation in reactive subjects.

The reagent withstood heating in the autoclave for thirty minutes at 120° C. with only a slight decrease in the reactions as compared with unheated fluid. Storage in the refrigerator for two to three weeks has no appreciable effect on the reagent.

These results indicate that all preparations containing an adequate concentration of virus particles are effective intradermal reagents; but in one experiment supernatant fluid from which much of the virus had been removed by high-speed centrifugation still produced a practically undiminished reaction.

Clinical Characteristics of the Reactions.

In some subjects, especially those volunteers repeatedly inoculated, a slight wheal surrounded by erythema develops about ten minutes after inoculation. This was also seen in some of the children inoculated with undiluted material. These reactions are apparently due to the chick protein and have been elicited with normal chick protein. So far we have had none sufficiently severe to cause any concern, though possible danger to allergic subjects must be kept in mind.

The site of inoculation sometimes becomes pale three or four hours after inoculation; but this bears no relation to the subsequent erythema. The erythema begins to appear at about eight to ten hours. The peak of the reaction is usually reached at between twenty-four and thirty hours, after which time it normally fades, so that often there is little left at forty-eight hours. Exceptionally it increases up to forty-eight hours. The reaction in children inoculated with undiluted fluid usually persists for forty-eight hours. The erythema is frequently accompanied by thickening of the skin and sometimes by itching. We read the reactions at twenty-four hours.

The same subjects usually give reactions whose diameter does not vary by more than $\pm$ 50% when tested on more than one occasion with the same reagent over a period of some weeks, provided the tests are done on a standard site, such as the anterior aspect of the forearm.

In two adults giving 20 millimetre reactions with the usual 1:10 dilution of influenza B fluid, dilutions of 1:100 and 1:1,000 respectively produced reactions about one-half and one-third as large. In five adults undiluted boiled influenza A and B fluids gave reactions 20 to 50 millimetres in diameter, deep pink in colour, slightly swollen and tender. Newcastle disease virus fluid undiluted and boiled, inoculated into two of these persons, produced reactions about half as large.

Repeated intradermal inoculations, and in one instance subcutaneous inoculation in addition, had no effect on subsequent reactions, providing the tests were made at different sites. However, if a second test is made at the same site three days after the first one, the reaction is partly or completely inhibited. This inhibition is equally effective against heterologous strains as against homologous strains. After three weeks a second inoculation at the same site may produce a normal reaction or an accelerated reaction coming up in about eight hours and fading by twenty-four hours.

The Reaction in Relation to Age and Influenzal Experience.

Age.

Adults Aged Twenty to Forty Years.—A series of 90 adults were inoculated with 0·1 millilitre of influenza A ("Melbourne"), influenza B ("Lee") and Newcastle disease virus reagents, prepared by dilution to 1:10 in saline solution and being boiled for ten minutes. Of the 180 tests with influenza A and B, the erythema produced in nine was from 21 to 30 millimetres in diameter, in 142 it was from 7·0 to 20 millimetres in diameter and in 24 it was less than 7·0 millimetres in diameter; no reaction was produced in five tests. In the case of the 90 tests with Newcastle disease virus, four reactions measured 11 to 15 millimetres in diameter, 17 measured from 7·0 to 10 millimetres in diameter and 31 measured from 3·0 to 6·0 millimetres in diameter; in 38 instances no reaction was produced.

Adolescents.—Eleven adolescents, aged from sixteen to twenty years, were tested with 1:10 dilutions of influenza A and B virus. In nine instances there were no reactions greater than 5·0 millimetres in diameter. When six of these people were tested with undiluted virus, they gave reactions from 10 millimetres to 30 millimetres in diameter.

Children.—Twenty-five children, aged from two to ten years, were inoculated with 1·10 dilutions of influenza A and B virus and Newcastle disease virus. Of the 75 possible reactions, only seven were 4·0 millimetres or more in diameter, and none were more than 10 millimetres in diameter. A further series of 24 children, aged two to twelve years, were inoculated with the same viruses undiluted. Of the 72 possible reactions, 12 were 10 to 20 millimetres in diameter and the remainder were smaller. Thus the reactions in children are much smaller than those in adults.

Relation of Degree of Reaction to Serum Antibody.

In over fifty adults, aged between twenty and forty years, there was no correlation between the degree of cutaneous reaction and the antibody titre as measured by the hæmagglutinin test. This was also the case with eleven adolescents aged sixteen to twenty years.

It is not surprising that in adults, all of whom almost certainly have been repeatedly infected by influenza viruses, there should be a lack of correlation between the degree of cutaneous reaction and the antibody titre, for there is no reason to believe that the circulating antibody as measured by neutralization tests *in vitro* or *in vivo* determines the skin reactions. However, in young children the position should be more clear-cut. A child with no detectable antibody has probably never suffered from influenza, while on the other hand one with detectable antibody must have suffered from influenza within some period less than his age—that is, relatively recently. Therefore it is of interest to study the relationship between the antibody titre and the cutaneous reaction in children.

Intradermal tests and serological investigations were carried out with 31 children. Full data on these are set out in Table I. It would seem permissible to regard reactions measuring less than 5·0 millimetres as probably of no significance. On this basis thirteen children yielded positive cutaneous reactions; data regarding these are summarized in Table II. In Table III the number of reactions against influenza virus A and influenza virus B are shown *vis-à-vis* the appropriate serological results.

Only two children reacted to Newcastle disease virus, and in both cases the reaction had completely faded at forty-eight hours, whereas nearly all the influenza reactions persisted for forty-eight hours.

The summarized data in Table III show that, of a total of eighteen cutaneous reactions, in seventeen instances the serological tests revealed that the child had suffered from the appropriate type of influenza. The one exception is the subject Law., who reacted to both A and B viruses, but had no antibody to B virus. On the other hand, there were many instances in which antibody was present but there was no cutaneous reactivity. Four children (Whi., Cop., Hin. and Rya.) reacted to A virus and not to B virus, and two (Bai., Kir.) reacted to B virus and not to

TABLE I.

Comparison of Cutaneous Reaction and Antibody Titre in Children.[1]

Name.	Age. (Years.)	Cutaneous Reactions in Millimetres.			Serological Tests.							
					Hæmagglutinin.		Neutralization in Mice.					
		A.	B.	C.	A.	B.	A.				B.	
		With Dilute Reagent (1:10).					*Serum+1:100 Virus.*					
Hin.	7	10	0	0	120	<10	0	0	0	0	3	3	2	0
Rya.	8	9	0	0	80	40	0	0	0	0	3	3	3	2
Mur.	10	4 (13)	1 (3)	0	120	30	0	0	0	0	0	0	0	0
Bla.	8	0	1	0	80	120	0	0	0	0	3	2	3	2
Tor.	9	3	2	0	30	100	0	0	0	0	0	0	0	0
San.	8	1	0	0	20	10	0	0	0	0	4'	3	3	3
McD.	5	4	1	2	10	<10	0	0	0	0	4'	3	1	0
Tur.	7	1	1	0			0	0	0	0		4	3	3
Web.	5	0	2	0	<10	<10	4s	4s	4s	3	2	2	2	1
		With Undiluted Reagent.			<10	80	4'	4'	4'	4'	0	0	0	0
					30	<10	0	0	0	0		4'	3	1
Kir.	9	2	7	2	60	25	0	0	0	0	1	0	0	0
Whi.	6	10	0	2	<10	<10	4'	4'	4'	4'	3	3	3	2
Lar.	6	15	11	0	<10	<10	0	0	0	0	3	2	2	1
Rob.	2	2	4	1	<10	<10	0	0	0	0	1	1	0	0
Wil.	4	0	2	0	<10	60	4'	4'	4'	3	3	3	2	2
Jen.	7	1	4	1										
And.	6	1	4	1			*Serum+1:10 Virus.*				*Serum+1:10 Virus.*			
Bai.	4	2	10	2	<10	40	4'	4'	4	3	0	0	0	0
Coop.	11	9	2	2	120	20	0	0	0	0	4s	4'	4	3
McN.	9	10	10	2	40	<10	0	0	0	0	2	1	1	0
Law.	5	9	7	1	15	15	0	0	0	0	4'	4'	4'	3
Coom.	12	12 (10)	12 (10)	12 (0)	20	40	0	0	0	0	0	0	0	0
War.	8	5 (10)	4 (6)	2	15	10		0	0	0	4s	4s	4s	4'
Sex.	7	15 (0)	17 (0)	20 (0)	30	20	0	0	0	0	3	3	2	2
McK.	5	0	2	3	<10	<10	4s	4s	4'	4'	4s	4s	4'	3
Cor.	12	0	2	0	30	15	0	0	0	0	2	2	0	0
For.	5	2	3	2	30	25	0	0	0	0	4s	4s	4s	4'
Mul.	8	3	3	0	30	10	0	0	0	0	4s	4s	3	3
Wal.	5	0	0	0	<10	20	4'	3	3	2	2	2	2	1
Obr.	9	3	3	3	40	30	0	0	0	0	2	2	1	0
Arc.	9	3	3	3	10	15	0	0	0	0		4s	4'	3
Cla.	6	2	3	3	15	10	0	0	0	0	4s	4s	4s	4'

[1] A = influenza strain "Melbourne", B = influenza strain "Lee", C = Newcastle disease virus. Cutaneous reactions were read at twenty-four hours; they were also examined at forty-eight hours, and when a significant change had occurred, this is recorded in parentheses after the twenty-four hour reading.

TABLE II.

Summarized Serological Data Concerning Children Showing Positive Skin Reactions.[1]

Name of Child.	Cutaneous Reactions.			Serological Tests.			
				A.		B.	
	A.	B.	C.	Mouse Test.	Hæmagglutinin	Mouse Test.	Hæmagglutinin.
Lar.	+	+	−	+	+	+	+
McN.	+	+	−	+	+	+	−
Com.	+	+	(+)	+	+	+	+
Sex.	(+)	(+)	(+)	+	+	+	+
Law.	+	+	−	+	+	+	+
Mur.	+	−	−	+	+	+	+
War.	+	±	−	+	+	−	−
Him.	+	−	−	+	+	−	−
Rya.	+	−	−	+	+	−	−
Whi.	+	−	−	+	+	−	−
Cop.	+	−	−	+	+	−	+
Kir...	−	+	−	−	−	+	+
Bal...	−	+	−	−	−	+	+

[1] Reactions in parentheses faded completely in forty-eight hours.

A virus; in each of these six cases the results of the serological tests corresponded completely with the cutaneous reactions.

TABLE III.

Correlation of Results of Serological Test with Cutaneous Reactivity in Children.

Virus.	Serological Test (Mouse).	Cutaneous Reaction.	No Cutaneous Reaction.
Influenza A.	Reaction	11	13
	No reaction ..	0	7
Influenza B.	Reaction	6	6
	No reaction ..	1	18

Discussion.

The main results of this investigation may be summarized as showing (i) that adults are more reactive than children, both to the specific and to the control preparations, and (ii) that in children a positive cutaneous reaction is indicative of past infection with the type of virus used for the test. Points calling for discussion are (i) the significance of the fairly high proportion of adult responses to Newcastle disease virus preparations, (ii) the evidence that influenza A and B virus types differ in their allergenic power, as in antigenic qualities generally, and (iii) the possible clinical or diagnostic significance of our demonstration that allergic phenomena may appear in response to influenza virus infections.

1. An adequate interpretation of the Newcastle disease virus reactions would require more extensive tests with control reagents on strong reactors than we have had any opportunity to carry out. Since on several occasions we have found that reactors to Newcastle disease virus did not respond to normal chick proteins, and since most reactors had never previously been inoculated with chick embryo materials, it is much more likely that the response is to be related to the virus component rather than to chick proteins. Since the possibility of past infection with Newcastle disease virus can be excluded, the simplest hypothesis is to assume a broadening of the specificity of the reactivity induced by past influenzal virus infections, so that a response is given to the distantly related Newcastle disease virus as well as to one or both of the influenza virus types. An alternative interpretation is that some product of cell damage produced by all three types of virus infection in the allantoic cavity, or conceivably the virus proteins themselves, have an intrinsically irritant effect on some adult skins. On at least three occasions we have observed an increase in reactivity to Newcastle disease virus in subjects infected with attenuated A and B virus and showing an increased cutaneous response to both A and B reagents as well. In two cases both influenza A and B antibody titres rose (as judged by hæmagglutinin tests); in the other only the influenza B antibody titre rose. This would favour the view that the Newcastle disease virus reaction is part of a "broad" allergic response to one or both types of influenza virus protein.

Caution is obviously needed in the interpretation of cutaneous reactions, especially such as these, which consist mainly of erythema and are usually fading by forty-eight hours. Individual measurements should not be taken too literally, because different skins vary in reactivity physiologically, because the erythema is sometimes diffuse and the limits are difficult to define, and because the measurements of diameter do not take into account the intensity of the erythema or the presence of absence of swelling.

Our general impression from work with these skin reactions in adults was that practically every subject was reactive to some degree, but that the varying occurrence of non-specific and semi-specific "broad" reactions made it quite impossible to relate the responses of a subject solely to his immunological history.

2. In children, on the contrary, non-specific reactions were rarely encountered. Specific reactions were much weaker; but when they occurred they were in all but one of eighteen instances associated with the corresponding type of circulating antibody, and could therefore be accepted as evidence of past infection with the virus concerned. Many children, however, were found to have antibody in the blood without corresponding skin reactivity. A sufficient number of children were found with evidence of infection with either influenza virus A or B only to make it clear that in children at least, the skin responses to the two types of antigen show the same distinct specificity as is observed in serological tests.

3. We have had no opportunity to study the course of skin reactivity in patients suffering from clinical influenza; but a considerable number of subjects being immunized by either intranasal or subcutaneous inoculations were tested intradermally before and three weeks after immunization. Very little correlation could be observed between the skin reactions and the immunological status of the subjects. The only significant feature observed was that a considerably greater proportion of subjects immunized with living virus given intranasally showed an increase in reactivity, as compared with those given formalin-killed virus subcutaneously. Details of these results will be reported in another paper.

In work on immunization with influenza virus B, Bull and Burnet[m] observed, particularly in one subject, that a second administration of attenuated virus might provoke more definite symptoms than the first given some months previously. Our present results make it appear likely

that many people, probably most adults, are to some extent allergic to influenza virus or its products. Taken together, the two groups of observations suggest that the allergic state may play some part in determining resistance or susceptibility to influenza. The allergic reactivity of the skin is presumably shared by the mucosa of the respiratory tract, and thus the allergic state of the subject may well play an important part, both in resistance and in the production of symptoms.

A full investigation of the reaction in patients suffering from influenza might provide some useful information on such points as the following: (*a*) whether the cutaneous reaction is specifically inhibited during the acute stage of influenza (if so, it may provide a diagnostic test in adults, nearly all of whom are normally reactors); (*b*) whether there is any relation between the degree of reaction and susceptibility to infection, or between the reaction and the severity of symptoms.

Summary.

1. Intradermal inoculation of a 1:10 dilution of unheated or boiled allantoic fluid infected with influenza virus A or B produces a cutaneous reaction in most adults and in some children.

2. Similar reactions are not produced by normal allantoic fluid or by suspensions of chick tissue containing from ten to twenty times as much protein as the influenza reagent.

3. Partial purification of the virus does not diminish its capacity to produce reactions.

4. In adults the size of the reaction bears no correlation to the serum antibody titre.

5. Among thirty-one children tested intradermally with influenza A and B reagents there were eighteen reactions, and in all but one instance the children were shown by serological test to have been infected in the past by the corresponding viruses. However, in many instances children who gave positive reactions to serological tests failed to give appropriate skin reactions.

6. The suggestion is made that allergy to the virus may play a part in resistance to infection, and when infection does occur, in the production of symptoms.

Acknowledgements.

We wish to thank Captain L. H. Bronson, of the United States Army Medical Corps, for assisting with the intradermal tests on adults, and Dr. A. G. Nicholson for assisting with the intradermal tests on children. All members of the staff of the virus department of this institute have cheerfully volunteered repeatedly to be subjected to intradermal inoculations. We are indebted to Miss B. Splatt for the protein analyses.

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Reports of Cases.

SOME CLINICAL NOTES ON MACROCYTIC ANÆMIA IN NEW GUINEA NATIVES.

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TRULY it is late in the day to be reporting cases of macrocytic anæmia in New Guinea natives, for their notes cover the twenty-year period from 1921 to 1941. They are written from the point of view of a casual clinical worker and from memory. There are few hæmatological data, no reticulocyte counts and no biochemical data. The object in seeking the clemency of the Editor is simply to record the fact that macrocytic anæmias exist amongst the primitive peoples of New Guinea. More thorough investigation of these cases by the hæmatologists in the future will no doubt throw light on their ætiology and pathology.

The diagnosis of the anæmia has largely rested on the rather typical blood picture seen in a thin blood film stained with Leishman stain; this shows pronounced anisocytosis, with inequality in the staining reactions of the red blood corpuscles. A large polychromatic red cell is a common feature. Megaloblasts could be demonstrated in every case—that is, large nucleated red blood corpuscles, about 16μ to 18μ in diameter, with a large, loosely textured nucleus, and a cytoplasm usually polychromatic, but sometimes eosinophilic (my apologies to T. E. Wilson[1] for this definition). In support of this simple diagnostic procedure, I should like to quote H. C. Trowell,[2] who makes the following statement in regard to his dimorphic anæmia (that is, nutritional macrocytic anæmia complicated by iron deficiency): "Dimorphic anæmia can, in the majority of cases, be diagnosed on the examination of a well stained and well spread blood smear. That is the only point the usual clinical worker should grasp."

Let me digress here for a moment to the blood picture seen in the early stages of blood regeneration in severe cases of blackwater fever in Europeans. The blood picture in the post-hæmoglobinuric phase resembled very closely the picture seen in the cases of macrocytic anæmia in natives. Pronounced anisocytosis was present, with many large polychromatic red blood corpuscles and big nucleated red cells. Erythropoiesis was disorderly and in part probably megaloblastic in type. After two or three weeks the anisocytosis disappeared and blood regeneration proceeded in normal fashion.

In support of this suggestion, Hamilton Fairley *et alii*,[3] in their preliminary report on nutritional macrocytic anæmia in Macedonia, under the heading of "Price Jones Curves in Blackwater Fever", make the following statement:

> In case 4 an Englishman, on the 8th day, had a megalocytosis of 9·2; reticulocytes which are known to be of larger diameter than ordinary red blood corpuscles equalled 10% at this the 8th day, but some 18 days later, when reticulocytes still equalled 10%, the megalocytosis had disappeared, and a normal Price Jones Curve had been re-established. It is a reasonable assumption that the megalocytosis was in this instance directly or indirectly related to hæmolysis, but not apparently to the reticulocyte response.

The macrocytic anæmia as seen in New Guinea natives is characterized by the following signs: (i) relatively acute onset with pyrexia, which may last weeks or months; (ii) rapid enlargement of the spleen, which is both painful and tender and diminishes in size during spontaneous remissions; (iii) the blood picture of a macrocytic anæmia; (iv) rapid loss of weight; (v) spontaneous remissions and relapses.

There is also a more chronic type of macrocytic anæmia, in which the spleen is enlarged, but not painful. The anæmia is of low grade and compatible with reasonably good health, and is amenable to liver therapy. It runs a non-febrile course. For some unknown reason the anæmia may at times become more active—as, for example, in the case of women during the later months of pregnancy. Case III is an example.

A factor of importance in the observation of these patients in general is the fact that they have been directly or indirectly under observation for a period of years. I have seen them both in the stage of relapse and in that of remission. About twenty cases were detected during the twenty years from 1921 to 1941. Most of the cases occurred among young adult males.

With regard to geographical distribution, the majority of patients resided in the south-eastern division of Papua with

Samarai as a centre, and were seen during the period from 1921 to 1927. One patient came from the Finschhafen area in New Guinea territory. In these areas malaria is hyperendemic, and the natives have developed full immunity. The adult is more or less free from attacks of malaria. Chronic malaria does not enter into the clinical picture. Strangely enough, no cases were detected during the period 1929 to 1936 in the dry coastal belt area with Port Moresby as a centre.

Case I.

A young indentured labourer from the D'Entrecasteaux group of islands was working on a coconut plantation in Milne Bay. He was under observation from 1921 to 1923. During this period he had three attacks of macrocytic anæmia accompanied by pyrexia and enlargement of the spleen. The issue was clouded in his first attack by the fact that he had a hookworm infestation. He received appropriate treatment for this and for malaria, as it was assumed that his anæmia was the result of a dual infection of malaria and hookworm. His clinical condition greatly improved after he had received this treatment in his first two attacks of macrocytic anæmia. During his third bout of anæmia he received no treatment of consequence. In spite of this, satisfactory blood regeneration took place, with diminution in the size of the spleen and subsidence of his fever. Malaria parasites were never at any time demonstrated in a thin blood film. As a tentative diagnosis pernicious anæmia was suggested. At that time, when we ate our butter out of tins and had no ice and no electric light, that was the only macrocytic anæmia of which I was cognizant. The patient was lost sight of after his third anæmic relapse.

Case II.

In 1923, a young village native from Maiwara village, Milne Bay, suffered from high fever, enlargement of the spleen, anæmia and emaciation. After fourteen days' treatment with arsenic, no improvement had occurred. At his own request he was allowed to return to his village. He was extremely anæmic and weak, with severe loss of weight, and his spleen was much enlarged. Four months later I had the good fortune to visit his village and found our patient restored to good health. He had no obvious anæmia, the spleen edge was just palpable below the left costal margin, he had no fever, and he had gained much weight.

Case III.

This patient was the young Milne Bay bride of our cook boy. She was first examined in 1924, when she appeared to be most unusually listless and dull. She was found to be suffering from a low-grade macrocytic anæmia, with a moderately enlarged spleen, but no fever. A great improvement in her general health and satisfactory blood regeneration took place during the period from 1924 to 1928; perhaps this was due to the fact that during this period she assisted her husband in the kitchen and had access to more good animal protein and fat than she would be likely to get in her village. In 1934 she became pregnant for the first time. At about the sixth month of her pregnancy a most violent exacerbation of her anæmia took place, and a most extraordinary enlargement of her spleen, which reached well down into the left iliac fossa and pushed the pregnant uterus well over to the right of the middle line. Circumstances prevented me from being present at the birth of her son at her village in Milne Bay. She must have been desperately anæmic at the time of her confinement, but with the help of her grandmother she survived the ordeal. In 1936 I learned that mother and infant were alive and well.

Case IV.

This case was detected at Samarai in 1927. The patient was seen before, during and after his attack of macrocytic anæmia. He was a young native from the D'Entrecasteaux group of islands. Two months prior to his attack of anæmia he was under treatment for gonorrhœa, and at that time he showed no signs of an abnormally large spleen. He gave no history of recent attacks of malaria. He was admitted to hospital on about the fifth day of his illness; his temperature was 104° F., his spleen was painful and tender and its edge was well below the umbilicus. The patient was breathless and weak and profoundly anæmic. The erythrocytes numbered 1,200,000 and the leucocytes were 5,000 per cubic millimetre. The blood picture was characterized by pronounced anisocytosis; megaloblasts were present. The sclerotics of his eyes were not jaundiced; bile pigment was not present in the urine; no urinary casts or cellular elements were detected in the centrifugalized urinary deposit. No malaria parasites were seen in a thin blood film. The heart and lungs appeared free from abnormality. On the eighth day of his illness the number of erythrocytes had fallen to 900,000 per cubic millimetre. The patient complained of dimness of vision. Ophthalmoscopic examination of his *fundus oculi* revealed retinal hæmorrhages. He became very restless and breathless. On the ninth day of his illness he was given a blood transfusion of 500 cubic centimetres of citrated blood; this was followed by an alarming rigor, with a rise of temperature to 106° F. On the tenth day the erythrocytes numbered 1,100,000 per cubic millimetre and his general condition had improved. For the next fourteen days the count of the number of erythrocytes remained more or less stationary, so that on the twenty-fourth day of his illness the number was 1,200,000 per cubic millimetre. This phase of the anæmia, during which there was little if any increase in the number of erythrocytes, coincided with the period of pyrexia; during the first two weeks of the anæmia his temperature ranged between 103° F. and 101° F., falling by lysis to normal by about the twenty-eighth day. From then on blood regeneration took place rapidly. On the ninetieth day the erythrocytes numbered 5,000,000 per cubic millimetre. The patient was completely restored to health, his eyesight and weight were normal and his spleen edge was palpable three fingers' width below the left costal margin. The boy, it is assumed, remained in good health up to the time of his death in 1933. His death by violence occurred in his native village.

Case V.

A corporal of the armed native constabulary was admitted to the Port Moresby Native Hospital in 1934, suffering from anæmia, macrocytic in type. His spleen was enlarged and tender, and pyrexia was present; the erythrocytes numbered 2,400,000 and the leucocytes 2,000 per cubic millimetre. Sudden death occurred at about the twenty-first day of his illness. The probable cause of death was a pulmonary embolus. *Post mortem*, the marrow in the shaft of the left femur was found to be red in colour.

Case VI.

A young indentured native from the Finschhafen area in New Guinea territory was seen at Wau in 1938. He had been ill for some three or four weeks prior to his admission to hospital, suffering from fever, weakness and loss of weight. On examination, his spleen was found to be enlarged, his temperature was 101° F., and he was weak and emaciated; the erythrocytes numbered 1,300,000 and the leucocytes 4,000 per cubic millimetre. The blood picture was one of macrocytic anæmia with many megaloblasts present. During his four weeks' stay in hospital his temperature varied between 100° F. and 98·4° F.

He received the following treatment. During the first week in hospital two blood transfusions of 500 cubic centimetres of citrated blood were given with some temporary benefit. During the second week four cubic centimetres of "Campolon" were given per day for seven days. During the third week, one ounce of "Marmite" per day was given for five days. Diarrhœa developed at that stage, and the "Marmite" was discontinued. During the fourth week, *Ferri et Ammonii Citras* was given in a dosage of two drachms per day for one week. On the twenty-eighth day of his stay in hospital the erythrocytes numbered 1,350,000 per cubic millimetre, and he had derived no benefit from his treatment. His spleen was not diminished in size. There was no change in the blood picture, nor had he gained in weight. The patient had not benefited from liver given parenterally in moderate doses. The patient went home to his village. Twelve months later, in 1939, I was informed by letter that the boy was in excellent health. It seemed reasonable to assume that on his return to his village a natural remission had occurred in his anæmic state, and regeneration of red blood corpuscles had been accomplished.

Acknowledgement.

I should like to thank Dr. A. H. Tebbutt for his advice in compiling the paper.

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The Medical Journal of Australia

SATURDAY, JANUARY 29, 1944.

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THE HEALTH OF GREAT BRITAIN.

A "SUMMARY REPORT" of the Ministry of Health of Great Britain for the year ended March 31, 1943, was presented by the Minister of Health to Parliament by command of His Majesty in September, 1943. The last two reports from the Ministry of Health of Great Britain have been examined in these pages with more than usual interest because in wartime the health of a nation is necessary to its victory; but apart from this the maintenance of health is dependent on so many factors that a good health report is reason for satisfaction and encouragement. The last two reports have left nothing to be desired in this regard, and of the present document much the same may be said. In the introductory letter to the present document the Right Honourable Ernest Brown, Minister of Health, states that in many ways the year under review has been remarkable. In the first place more and more attention has been given to post-war planning. In this regard local authorities have been urged to lay down a housing programme for "at least the first year of the peace"; hospital surveys have been made; progress has been made in inquiries into the organization and distribution of medical teaching and research facilities; and an inquiry has been undertaken into the provision of dental services. Preliminary discussions have also been begun with the interests concerned in Assumption B of the Beveridge Report. In the second place attention has been paid to current progress and reform, and four aspects receive special mention under this heading. The building of new houses has been resumed; steps have been taken to protect the rights, increase the pay and improve the status of nurses; new methods have been adopted in the attack on the "two great social scourges" of venereal disease and pulmonary tuberculosis. In addition to all this the year produced a series of favourable records in vital statistics. The report itself is divided into five chapters, and these deal with: "The Public Health", "The Year's Work in Other Fields", "Mothers and Children", "The Problem of Housing", and "Specialist Medical Work in the Emergency Hospital Scheme". No attempt will be made to summarize all the information in these chapters—the report itself is only a "summary report"—but an attempt will be made to show that Great Britain is not only maintaining the health of its people, but doing so with an eye to the future.

From time to time during the year under review it has been stated that though venereal diseases and tuberculosis were black spots, the general health of the nation continued to be good and in some respects even better than in peace time. The statistics for the full year show "beyond doubt" that this statement was well founded. The year 1942 was one of record breaking in vital statistics. Not only the maternal and infant mortality rates, but also the proportion of stillbirths, and the standardized death rates among civilians, both male and female, were the lowest ever recorded in England and Wales. The incidence of infectious diseases was probably the lowest on record. It is pointed out that though statistics provide a basis for a strict comparison with the years of peace, and give reliable evidence of the general well-being of the population, they cannot reflect the health situation in detail or measure "positive health"; further, minor ailments and the "below par" or "tired feeling" do not come within their scope. There is no way of measuring the health of forty million people. Thus a number of nutritional surveys made in 1942 showed that there was no general deterioration in the nutritional state of the people. On the other hand, information supplied by doctors, together with the increased number of claims for sickness benefit under the national health insurance scheme, suggested that there was a considerable increase in short-term sickness during the year. It is difficult to determine the significance of these observations. Several important considerations are put forward as showing the need for caution. The first suggestion is most interesting—as a result of the teaching about "positive health" and preventive medicine, doctors have carried their activities beyond the bounds of curative medicine and ordinary people are beginning to realize that they can enjoy "health with a polish on it". This means that people have become increasingly aware of how much illness still exists. Possibly also people are seeking medical advice more regularly and are seeking it more generally in the earlier stages of illness. It would be strange if this were not so in view of all the modern clinics—ante-natal clinics, child welfare clinics, school medical services and so on. During war also medical inspections and examinations are undertaken much more often than in peace time, and people in increasing numbers are no doubt overcoming any fears that they may have had of such procedures. Crowded consulting rooms do not necessarily indicate an increased amount of illness. In this connexion several statements are made. On the one hand fewer doctors are available to the civilian population and there appear to have been more visits to surgeries and fewer visits by doctors to the patients' homes. On the other hand "it is only to be expected that fatigue arising from the tempo and strains of life in wartime should often reduce the feeling of well-being, and may sometimes convert latent into subacute disease or in some instances protract recovery from minor illness". It should be remarked in passing that many of these statements would be just as true of Australia as they are of the Old Country.

Turning to some of the details of the vital statistics, we find that the total number of deaths in England and Wales in 1942, including non-civilian deaths registered in

the country and those due to enemy action, was 480,137, or 55,043 less than in 1941, the general death rate among the civilian population being "remarkably low". Among females the standardized death rate was 6·84 per thousand living, or 8% better than in any previous year, notwithstanding the inclusion of deaths from enemy action and the withdrawal of large numbers of healthy young women from the civilian population. Among males the standardized death rate was 9·52‰ and the lowest on record; this was in spite of the "considerable effect of selective recruitment". Mortality of children at the pre-school ages one to five years, which had declined by no less than 47% between 1931-1935 and 1939, showed a further improvement of 2% in 1942; and at the school ages of five to fifteen years "the low level reached in 1939 was regained". The total number of live births (654,039) was greater than the total in 1941 by 66,811; taken with the total number of deaths registered in England and Wales, this gives a natural increase during 1942 of 173,902. The rate of 15·8‰ of the mid-1939 population was the highest since 1931. The crude infant mortality rate of 49 per thousand live births registered in the same year is the lowest on record, and the same is true of the stillbirth rate of 33 per thousand total births. The maternal mortality reached the record low level of 2·47‰. A list of deaths from various diseases is set out and alongside is given the previous lowest annual number recorded since 1930. The statement is made that without doubt sulphonamide therapy has been responsible in large measure for the decline since 1938 in deaths from a number of the causes mentioned. The principal diseases which caused an increased number of deaths as compared with 1941 were cancer, leuchæmia, Hodgkin's disease, cerebral embolism and thrombosis, coronary disease and *angina pectoris*, enteritis and diarrhœa, hepatitis, certain diseases of the genital organs and osteomyelitis. The low incidence of infectious diseases has already been mentioned. Together with this most encouraging picture of the reduction in the incidence of, and in deaths from, certain diseases, there is a realization that much still remains to be done among certain persons in the teaching of simple rules of health. A survey was made by the Ministry of Information for the Ministry of Health, and we are told that surprising ignorance on health matters still exists even among those who have not the excuse of poverty, over-crowding or inadequate facilities. It is not only among what has been described as "the submerged tenth" that lack of cleanliness is noticed.

Special mention has been made of venereal diseases and tuberculosis. The number of civilian sufferers from syphilis who attended treatment centres for the first time in 1942 was 9,046. With service cases added this represented an increase of 29·6%, compared with 1941; there was a rise of 40% between 1940 and 1941. The increase in new syphilitic infections since the beginning of the war now amounts to about 120%. The incidence of gonorrhœa cannot be so easily estimated. The introduction of Regulation 33B was an attempt to deal with the problem of venereal disease. This regulation "made it an offence for any person indicted as a source of their infection by at least two patients under treatment for venereal disease to refuse examination or, if necessary, treatment by a special practitioner after being required to do so by the medical officer of health of a county or county borough, or to cease treatment until certified as not suffering from the disease in a communicable form". This regulation was discussed in these pages in the issue of October 9, 1943, and reference was also made to the Press campaign in England for publicity regarding venereal diseases. The subject has been dealt with in broadcasts and a film pleading for free and frank discussion of the problem was shown in every cinema in Great Britain under the auspices of the Ministry of Information. Although tuberculosis is described as one of the dark spots in the health picture, "the position in the third year of war was better than could reasonably have been expected two years ago". A wartime increase in the death rate occurred, but there has been a "distinct check" to this increase. A rise of about 3% in the number of new cases occurred during the year, and "under the trying conditions of total war it is not unlikely that the death rate will tend to rise again". Two important steps that will have effects in the future have been taken. Mass miniature radiography has been introduced, and a scheme has been initiated for the payment of special allowances to sufferers undergoing treatment in order that their dependants may not suffer hardship.

So far we have dealt with the chief features in the first of the five "chapters" of this report, the chapter in which the general results of the year are described. The other four chapters are not less important, and though they describe many activities that will be beneficial in the future, they are not so important for our present purpose. It will suffice if the chief aspects are mentioned. Mention is made of a "new deal" for nurses. A Nurses Salaries Committee was appointed and made recommendations on conditions of service, training and so on. The recommendations were commended by the Minister to all authorities. The *Nurses Act*, 1943, has been introduced and has three objects—to give a proper status and recognition to the grade of "assistant nurse" by the setting up of a roll under the control of the General Nursing Council, to protect the general public by restricting the use of the title "nurse" to persons with some nursing qualification, and to bring the agencies for the supply of nurses under control. In addition special machinery has been set up under the Ministry of Labour and National Service to deal with the continued shortage of nurses and midwives and their uneven distribution. In the previous reports the special consideration shown to expectant and nursing mothers and babies in the matter of food and food supplements was described. The arrangements during the year under review have been extended. Further wartime nurseries have been opened. During the year the number of whole-time nurseries increased from 712 to 1,489 and the number of part-time nurseries decreased from 206 to 142. Courses of training for the staffs of wartime nurseries have been extended. The number of residential nurseries has been increased; 52 nurseries with 1,600 cots were added in 1942-1943. There are now 193 hostels of different types for aged people who are either evacuees or homeless. A whole chapter is devoted to the problem of housing. A summary of this section cannot be attempted; it can, however, be stated that a serious attempt is being made to deal with the needs of the situation. The final chapter dealing with the emergency hospital scheme can only be mentioned.

This summary report, which can be obtained from His Majesty's Stationery Office for the sum of one shilling, merits careful study.

Current Comment.

A NEW TREATMENT FOR THYREOTOXICOSIS.

IN May of last year, E. B. Astwood, in an important contribution from the Harvard Medical School and the Peter Bent Brigham Hospital,[1] reported investigations in the treatment of patients suffering from hyperthyreoidism. He showed that two series of chemical compounds had the property of inhibiting the endocrine function of the thyreoid gland. When these compounds were given to animals there followed after a short latent period a lowering of the basal oxygen consumption, a decrease in the rate of growth and development, and a diminished intake of food—changes which, he indicated, were consistent with a state of hypothyreoidism. In some animals these changes are accompanied by a hyperplasia of the thyreoid gland "which is apparently compensatory in nature and mediated by the anterior lobe of the pituitary". In this regard Astwood quoted work by J. B. and C. G. Mackenzie and E. V. McCollum on sulphaguanidine, and by C. P. Richter and K. H. Clisby, and by T. H. Kennedy on thiourea derivatives. Further studies, he states, have shown that the primary action of compounds of these two types is an inhibiting action on thyreoid hormone production. Astwood tested more than one hundred compounds in order to select for clinical use highly active substances of low toxicity. Most of the derivatives of thiourea exhibited some activity, but varied widely in toxicity; thiourea itself was the least toxic of all. From the clinical point of view thiourea and thiouracil seemed to be the most promising and were used in studies on normal persons and on patients suffering from hyperthyreoidism. Astwood gives clinical histories of three patients and concludes that the daily administration of 1·0 to 2·0 grammes of thiourea or of 0·2 to 1·0 gramme of thiouracil to persons suffering from hyperthyreoidism brings about relief of symptoms and return to normal of the serum cholesterol level and of the basal metabolic rate. He recorded the occurrence of a latent period of from one to two weeks before these effects occurred; the remission was sustained while treatment lasted and the hyperthyreoidism returned when the therapy was discontinued. Astwood regarded his studies as preliminary only, and insisted that observation of a large number of cases over a long period of time would be necessary before the true merits and dangers of such a form of treatment could be determined.

What is stated to be the full confirmation of all Astwood's claims comes from H. P. Himsworth, Professor of Medicine in the University of London.[2] Himsworth points out that available results are so striking as to indicate that in thiourea and thiouracil clinicians have obtained for the treatment of thyreotoxicosis therapeutic agents that are superior to any so far available. He has used thiourea in six cases and gives the clinical history of the result in one of the most severe. The result, to say the least of it, was dramatic. On admission to hospital the patient presented the classical signs of severe Graves's disease; the pulse rate was 150 in the minute and the basal metabolic rate was +81% and +82% on two occasions. On the eleventh day of thiourea administration improvement began to occur; it occurred so rapidly that by the thirty-third day a casual observer would not have noticed that the patient had thyreotoxicosis. "His whole manner had changed; he was calm, collected and deliberate in his movements. The flush had left his skin; he no longer sweated and even the palms of his hands were dry. The tremor had disappeared completely. The staring eyes were no longer obvious. The lid retraction had gone, as also had the conjunctival injection; but on closer inspection it could be seen that the degree of exophthalmos was not significantly less." The goitre was softer, but it had not decreased in size; indeed measurement of the neck suggested that it was a little larger. The basal metabolic rate was +23% and +21% on the thirty-first and thirty-second days respectively. Himsworth describes two minor disadvantages of the treatment. The first is that thiourea has a nauseating taste and may cause vomiting; the second is that it imparts a peculiar sweet smell to the breath—more unpleasant to the observer than to the patient. Thiouracil has not been available to Himsworth. Since the dosage advised by Astwood is smaller than that of thiourea and its taste is "much pleasanter", Himsworth thinks that it may prove to be the better drug to use.

The reports of Astwood and of Himsworth are stimulating and cannot fail to fire the imagination. It may be, as one Australian authority has suggested, that before long surgical operation in thyreotoxicosis will be outmoded; but caution is necessary. This work is still to be regarded as in the experimental stage. So far records of more than a few months' treatment have not been published. We do not know whether the thiourea (or thiouracil) will have to be given for an indefinite period and we have yet to discover whether prolonged administration will have any untoward side-effects. Further, though the drugs in question appear to produce their effect by interfering with the synthesis of the thyreoid hormone, it is undoubted that more remains to be learned about this aspect. The treatment will be adopted in Australia—already a series of case histories are being prepared for submission to this journal—and it is necessary that full and complete records of all clinical and laboratory findings be kept. Particular care must be taken that every patient so treated is followed up with unremitting zeal by a competent observer.

[1] *The Journal of the American Medical Association*, May 8, 1943.
[2] *The Lancet*, October 16, 1943.

SNORING AND ITS PREVENTION.

PEOPLE who snore are not often distressful to themselves, but they can be a great nuisance to those with whom they live. In a preliminary report[1] on "a new approach" to the treatment of snoring, Jerome F. Strauss describes snoring as a seriocomic symptom and adds that it is often treated more lightly than it deserves, "as will be attested by many whose domestic tranquillity may be strained to the breaking point by it". The point is that a competent and frequent snorer will need to be convinced of his "nuisance value" and have sympathy with those afflicted by him if he is to undergo treatment that will cause him any inconvenience. Strauss insists that a proper interpretation and definition of snoring are necessary antecedents to an intelligent attack on it. All noisy inspiratory and expiratory respiration of a sleeping person is not snoring—laryngeal stridor, nocturnal groans, asthmatic wheezing and the noise caused by coarse râles are among the sounds that have to be excluded. True snoring is "a coarse low-pitched noise produced by vibrating soft tissue in the nasopharynx of a sleeping person". Strauss describes organic snorers and functional snorers. Organic snoring is dependent on such conditions as the presence of adenoids, a tumour of the naso-pharynx, a defection of the septum, polypi and nasal allergic states. Functional snorers have an essentially normal nose and throat; some of them have had lesions removed or deformities corrected. The sound in snoring is produced by the vibration of soft tissue. Each soft palate and uvula has its own "flutter ratio", and Strauss's idea is that if the flutter ratio can be altered by the use of sclerosing substances modifying the weight density and flexibility of the vibrating tissues, snoring will be reduced or eliminated. He has used "Sylnasol" (a 5% solution of the sodium salts of certain of the fatty acids of the oil extracted from a seed of the psyllium group). This substance is injected into the body of the soft palate, base of the uvula and free edges of the posterior pillars of the fauces. Not more than 0·5 cubic centimetre is injected at one sitting, and injections are given at intervals of a week for a period of five or six weeks. Some transient otalgia results; œdema is prevented by smallness in the amount of fluid injected. Strauss gives histories of seven patients treated, four of them with success. Further work along these lines will be welcomed.

[1] *Archives of Otolaryngology*, September, 1943.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Intraocular Injection of Sulphanilamide in Purulent Iridocyclitis.

J. IGERSHEIMER (*American Journal of Ophthalmology*, October, 1943) records what he believes to be the first case in which a sulphonamide has been injected into the anterior chamber of the eye. The patient was a boy, aged twelve years, whose eye had been injured two days previously; it had been struck by glass as a result of the explosion of a bottle of water. Vision in the eye amounted to light perception only, and an irregular central laceration of the cornea was present. The anterior chamber was formed, and a hypopyon was present in its lower quarter. There was a thick, fibrous exudate extending from the posterior surface of the cornea to the pupillary opening. The pupil was contracted and the iris congested, and the fundus could not be seen. Sulphathiazole was administered orally and 3,500,000 units of typhoid vaccine were given by intravenous injection, whilst atropine and adrenaline were applied locally. Some improvement occurred, but the aqueous remained cloudy. During the child's stay in hospital light perception almost disappeared. Seventeen days after the injury the condition of the eye was such that enucleation was planned; but the author decided that in such a "lost case" it was justifiable to risk injections of sulphanilamide into the anterior chamber. Accordingly about 0·2 cubic centimetre of aqueous was removed, and a 0·8% (saturated) solution of sulphanilamide was injected; the injection was well tolerated. Two days later a second injection was given. Six days after the first injection the condition of the eye was improved, and eight days later still iridectomy was possible. Five days after the operation the eye was white and quiet, and light perception had returned. Three months later a second iridectomy was performed, and a cataractous lens became visible; this was removed five months later again. The final result was a quiet condition of the eye and visual acuity of $^{20}/_{\infty}$. The author does not advise the frequent use of this procedure; but he considers that it may be tried in special cases. He describes in detail the technique of injection of sulphanilamide into the anterior chamber.

Iontotherapy.

NORMAN FLEMING (*The British Journal of Ophthalmology*, August, 1943) states that iontotherapy is treatment by ions electrically administered. The electrical apparatus employed should derive its power from small batteries, and the milliamperage used should never exceed two. Experience with weak solutions will rapidly remove all temptation to use stronger ones. The author believes that this method of introducing drugs offers unique assistance in the treatment of all kinds of ophthalmic conditions; but he does not advocate that it should replace any of the tried and trusted methods. He stresses the fact that the satisfactory treatment of all secondary inflammations still depends upon discovery of the cause; but that does not reduce the value of empirical treatment during investigation, or of iontotherapy in clearing up inflammation of which the cause has been eradicated. He has over a period of ten years treated about a thousand patients, and he reports a number of illustrative cases. Considerable success has been achieved, even in the treatment of such deep inflammations as scleritis, iridocyclitis and retrobulbar neuritis. The author's favourite application is six parts of calcium chloride (1 in 500) with one part of adrenaline hydrochloride (1 in 10,000), the actual strength of adrenaline used being 1 in 70,000; but he has been able to obtain an adrenaline effect with a dilution of 1 in 250,000. Only rarely does he apply treatment directly to the eye for more than two minutes all told, to the everted lids for one and a half minutes, and to the closed lids for five minutes. He uses calcium externally in nearly every case, adrenaline in all cases in which it is not contraindicated, zinc sulphate when epithelium has been lost or the mucous membrane is indolent, silver nitrate or "Prontosil Soluble" when a powerful antiseptic is required, atropine sulphate given in this manner as an alternative to its ordinary therapeutic use, acetylcholine and histamine to promote vasodilatation and for other purposes. Many other drugs may be used; but a few serve as well as many, and those one knows better than those which one does not. Treatment should be varied both according to the case and in the treatment of the individual patient. Treatments should be sufficiently spaced, since the results are then more easily observed. It is seldom necessary to see the patient often; but on rare occasions daily treatment is indispensable. The author believes that iontotherapy will be of particular value in the treatment of war casualties.

Ocular Manifestations of Spontaneous Subarachnoid Hæmorrhage.

A. J. BALLANTYNE (*The British Journal of Ophthalmology*, September, 1943) presents five cases of non-traumatic subarachnoid hæmorrhage, and discusses the pathological findings at post-mortem examination. He states that the discovery of hæmorrhage in the subarachnoid space at the base of the brain, within the sheaths of the optic nerve, and in many cases in the retina and even the vitreous, originally led to the belief that the blood passed in a continuous manner in the subarachnoid space through the optic canal into the orbit, and thence either along the course of the central retinal vessels or by lymph channels accompanying the optic nerve at the periphery of the *lamina cribrosa* into the retina. It is now generally held that the blood in the cerebral subarachnoid space does not, as a rule, travel through the optic foramen into the subarachnoid space of the optic nerve. It is thought that hæmorrhage occurs in the dural sheath at the apex of the orbit, and tracks forward into the subdural and subarachnoid spaces, and that blood in the last-mentioned position, by causing pressure and tension on the central retinal vein where it crosses the space, induces stasis in the central retinal vein, and thus accounts for the retinal and vitreous hæmorrhages. From investigation of his cases, the author states that hæmorrhages occur over a much wider field than the optic nerve sheaths, the retina and the vitreous, and that these hæmorrhages cannot be explained by the assumption of pressure on the central retinal vein. The hæmorrhages varied in distribution in the different cases; but they were found in and between the sheaths of the optic nerve, in the orbital fat and among the orbital muscles, surrounding the posterior ciliary nerves and the ciliary ganglion, the ophthalmic and posterior ciliary arteries and the vessels of the chiasma and optic tracts, as well as beneath the retina, in all its layers, in front of the retina and in the vitreous. The hæmorrhages were discrete and independent, and simultaneous in occurrence. The author submits that the occurrence of such multiple hæmorrhages can be explained only by a sudden rise of intracranial pressure causing stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit. He suggests that some of the clinical signs of subarachnoid hæmorrhage, such as oculomotor pareses and disturbances of conjugate movements of the eyes, may be explained by the occurrence in the mid-brain of hæmorrhages similar to those found in one of his cases in the chiasma and optic tracts.

Subhyaloid Hæmorrhage following Inoculation with "T.A.B." Vaccine.

J. P. F. LLOYD (*The British Journal of Ophthalmology*, October, 1943) states that it is well known that spontaneous hæmorrhages may occur in any part of the body after routine immunization with "T.A.B." vaccine. He records what he believes to be the first case of such hæmorrhage occurring in the eye. The patient was a man, aged thirty-six years, who was given two routine injections of the vaccine, the second fifteen days after the first; both produced normal reactions. The day after the second injection he noticed that the visual acuity of his left eye was poor; but he did not report for medical attention until some days later. When he was examined the left eye could discern hand movements only, and an extensive subhyaloid hæmorrhage was found to have occurred in front of the macular region, to the temporal side. He was put completely at rest and atropine drops were instilled. Absorption of the hæmorrhage began, but was slow. Seventeen days after treatment was begun visual acuity was still that of perception of hand movements only, and approximately two months later considerable unabsorbed blood was still present.

Herpes Zoster Ophthalmicus.

T. G. WYNNE PARRY AND G. C. LASZLO (*The British Journal of Ophthalmology*, October, 1943) record two cases in which ophthalmic disorders followed *herpes zoster*. The first patient had had an attack of *herpes zoster* along the ophthalmic branch of the fifth nerve six weeks prior to her admission to hospital; the cornea was not involved, but she had skin eruptions with severe pain, and three and a half weeks after the eruption she completely and suddenly lost the sight of her right eye. At the time of her admission to hospital she had perception of light at

three metres, and powers of projection in the periphery of the field, but none centrally. A diagnosis of acute bulbar neuritis was made. The second patient had had "shingles" on his chest six weeks prior to examination, and three weeks later he felt giddy and had double vision. Visual acuity was normal, the visual fields were full, and both disks were normal. The pupils reacted promptly, and no blepharoptosis or obvious squint was present. The right eyeball moved somewhat slowly on looking to the right, but ran the whole course. Right-sided homonymous double images were found by means of red and green glasses. A diagnosis of right abducent nerve paresis was made. In both cases full investigation revealed that the ætiology of the eye condition was *herpes zoster*.

Anisocycloplegia.

S. JUDD BEACH (*American Journal of Ophthalmology*, May, 1943) states that the term "anisocycloplegia" is coined to describe a caprice of cycloplegia, such that one eye of a subject is affected by a mydriatic drug to a greater extent than its fellow. Different methods of administration of the drug do not appreciably or consistently alter the response. The phenomenon appears to be a characteristic of the individual eye. It may be deceptive to any examiner taught to rely entirely upon cycloplegics; but it is a menace chiefly to those who usually prescribe on their unverified cycloplegic or retinoscopic findings. It is revealed by tests for depth of cycloplegia and by comparison with results of non-cycloplegic procedures. In a series of over 100 cases studied by the author, in seventeen there was a difference of over half a diopter in depth of cycloplegia in the two eyes.

OTO-RHINO-LARYNGOLOGY.

Penicillin and Tyrothricin in Otolaryngology.

S. J. CROWE, A. M. FISHER, A. T. WARD AND M. K. FOLEY (*Annals of Otology, Rhinology and Laryngology*, September, 1943) discuss the nature and properties of the two antibacterial agents, penicillin and tyrothricin. Penicillin is unstable and loses its potency rather rapidly. If it is kept in the ordinary ice box in open flasks, antibacterial activity is retained for only one week, while if similarly stored, but in rubber-capped bottles, potency lasts about one month. If frozen until required for use and then refrozen, it may be kept from two to five months. Penicillin is readily soluble and exhibits no toxic nor irritant effects, and thus in purified form may be administered intravenously or subcutaneously, or may be applied directly to infected areas or raw tissues. In high concentration it is bactericidal, while in greater dilutions it is bacteriostatic. Activity is not destroyed by pus or exudates, nor by para-amino benzoic acid. Tyrothricin is obtained from peptone cultures of the aerobic soil bacterium, *Bacillus brevis*. It is an impure substance containing gramicidin, an agent which is bactericidal for Gram-positive organisms only. Tyrothricin is insoluble in water, but dissolves readily in alcohol. The drug is stored in alcoholic solution and remains potent for considerable periods. It loses potency soon after contact with water and is precipitated by even minute quantities of mineral matter in water. For use, solutions are made when required, doubly distilled water being used to provide suspensions of 1 in 5,000 to 1 in 20,000. Tyrothricin causes hæmolysis and is thus unsuitable for injection, and, although slightly irritating to mucous membrane, it is not destructive to tissues, neither is the drug absorbed from the membranes or raw surfaces. Tyrothricin is rapidly bactericidal, but needs to be enabled to make direct contact with the bacteria. Its value appears to be great when employed to supplement surgical procedures such as the operations for chronic sinusitis and mastoiditis; it is of little use when merely used as nose or ear drops or in acute or chronic infections in which operation has not been performed. Both substances are highly selective in their action upon bacteria, so that the authors stress the need in every case for quantitative susceptibility tests in the laboratory and the use of cultures of the bacteria isolated from the disease regions. In this regard it is emphasized that anaerobic pathogens are not uncommonly found in nose and ear disease and that appropriate culture methods should be included in investigations for ætiological agents and subsequent susceptibility tests. Both agents are effective against Gram-positive bacteria. Streptococci are more resistant than pneumococci and staphylococci of all the Gram-positive organisms are the most resistant, although some appear to be sensitive. Also there are variations in the resistance of different strains of the same bacterial group. One may be sensitive to tyrothricin and insensitive to penicillin and *vice versa*. Tables summarizing the results of *in vitro* sensitivity tests clearly reveal the variable degree of control of growth upon different strains of *Staphylococcus aureus*. Penicillin appears to be the more potent against *Staphylococcus albus*. Studies with hæmolytic streptococci reveal that the Lancefield B and D forms, both of which resist sulphonamides, are sensitive to tyrothricin and to penicillin respectively. In cultures and in clinical records, the lethal effect of both drugs on pneumococci is evident. Over a period of two years tyrothricin and an unrefined pyrogen-containing penicillin have been employed at Johns Hopkins Hospital for local application in ear, nose and throat conditions. A study of 118 cases of *otitis media*, mastoiditis and sinusitis so treated forms the basis of this report. It is stated that the results obtained were better than had ever before been experienced. A tabulated series of a number of the cases reveals some very impressive results when the organism was shown to be sensitive, contrasting with the more obstinate course and more radical surgical procedures required in those cases in which *in vitro* tests revealed an insensitive infective agent. The value of tyrothricin as a local application in the form of post-operative packing and later as lavage is strikingly revealed. Conservative operations on the frontal sinuses, such as a small exposure and partial cleaning out, appears often to suffice when this agent is able to be employed and the organisms are sensitive to it. After radical sinus operations tyrothricin appears to be able to check and prevent infection, so that during healing the sinus remains free of pus and convalescence is shortened. Not much benefit was obtained in chronic states from the use of solutions of the drugs merely as drops or for douching without operative exposure, although some reduction in bacterial concentration appeared to result. Of the two agents, penicillin seems to be the more useful in acute conditions, probably by reason of its ready solubility and ability to penetrate infected tissues. Superiority over sulphonamides also is due to absence of any tendency to clog or form crusts and to the fact that no dangerous sensitized state is produced by local application, as is occasionally an unpredictable result of the use of the sulphonamides.

Rhinoliths.

C. J. POLSON (*The Journal of Laryngology and Otology*, March, 1943) records details of information derived from the study of 384 rhinoliths reported in the literature during the past 98 years. Foreign bodies apparently form the nucleus around which the stone develops in most, if not in all, cases. Of these, exogenous objects such as cherry stones, seeds, buttons, pieces of paper, are the commonest. Endogenous particles such as a tooth, a bony sequestrum or blood clot appear to have formed the nucleus in other instances. Although a foreign body is the principal factor which predisposes to rhinolith formation, it is not of itself a sufficient cause, for stone formation does not always take place about a foreign body, even after many years, and in the antrum encrustation is rare. Pus formation, with stasis and concentration beyond the point of solubility produced by the action of air currents so that precipitation occurs, appear to be events of likely influence. The principal minerals found are calcium phosphate and magnesium phosphate and traces of carbonates. The origin of calcium salts has been debated. Whereas tears and nasal mucus contain traces of calcium, pus may contain up to 30% of calcium by dry weight. Suppuration is thus considered to be an important influence in the development of a rhinolith around a foreign body. Unilateral nasal obstruction and discharge are the usual symptoms. Epistaxis, headaches, epiphora, and swelling of the nose or face are less common. The duration of stay of the foreign body and the period of symptoms are variable. A foreign body may remain for a long time and produce few or no symptoms. In the series studied, retention for as long as sixty years was reported and in a large number of cases symptoms were tolerated for many years, the explanation being the frequently low mentality of the patient prone to be affected. Diagnosis is usually made on examination with simple instruments, especially if the probe is used. X-ray examination may at times be useful. Complications do not appear to be common. *Rhinitis caseosa* and cholesteatoma were found in a small proportion of the cases, while sinusitis and *otitis media* were rare. Prompt relief of symptoms followed removal of the concretion in almost all the cases, and permanent structural damage was found to be rare. Occasionally rather severe hæmorrhage was encountered during removal.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on August 26, 1943, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. K. S. M. BROWN, the President, in the chair.

Nutritional Deficiencies.

SURGEON LIEUTENANT-COMMANDER J. K. MADDOX read a paper entitled "Remarks Concerning Vitamin Deficiency in the Australian Adult" (see page 81).

DR. RAYMOND GREEN, in a second paper, said that the deficiency diseases affecting children in Australia were rickets and scurvy. Infantile beriberi and pellagra were not seen and did not differ in their clinical manifestations from the adult type. Rickets was a disease of the rapid growth period of early childhood; it was the result of general body changes brought about by lack of the fat-soluble vitamin *D*. The most obvious changes were in the bony skeleton, and were due to interference with the calcium-phosphorus metabolism, which for its normal working required the vitamin in adequate amounts. There might be some lowering of the calcium level in the blood, though this was not common; if it was present, it might be accompanied by one of the spasmophilic group of diseases. The commoner type was that in which the blood phosphorus level was below the normal four to six milligrammes per 100 cubic centimetres. The ferment, phosphatase, necessary for the utilization of the phosphorus in bone-building, was not reduced. The phosphorus blood level was lowered by excessive loss in the urine. Dr. Green then quoted a statement from the latest edition of Sheldon's "Diseases of Infancy and Childhood": "Increasing knowledge has made rickets one of the most easily preventable diseases, with the result that it is rapidly becoming an uncommon condition and, in fact, the gross skeletal deformities are already a rarity." Dr. Green said that while the gross deformities were rarely seen in Australia, it was impossible to say that rickets was rapidly disappearing. It was, unfortunately, all too common in New South Wales; it was certainly mild, but quite recognizable.

Dr. Green went on to say that rickets was a disease of gradual onset, and signs were not apparent before the fourth month, and more commonly from the sixth month onwards. The infants looked healthy enough at first sight, but were flabby and at times restless and irritable. Excessive sweating of the head, poor appetite, delay in sitting up, late dentition and a tendency to catarrhal conditions (colds, bronchitis and diarrhœa) were present. Convulsions might occur from causes much slighter than usual. One of the signs in the infant was squareness of the head (bossing of the frontal bones with the coronal suture felt as a groove, the sagittal suture being less affected, so that one did not see the "hot-cross bun" head); the occiput was flattened, owing to the fact that the babies were sluggish and tended to lie a lot instead of sitting, so that the prolonged pressure of the pillow deformed the soft skull bones. Baldness of the occiput might often be seen from the constant rubbing of the head on the pillow. The anterior fontanelle would be found widely open, and the posterior fontanelle might be palpated up till the ninth month. A further effect was often found in narrowness of the upper jaw with a high-arched palate, while the lower jaw was poorly developed. Dentition was delayed, and the normal order of eruption of the teeth was often upset. Craniotabes did not occur as a rhachitic manifestation in Australia. In the chest one noted beading of the costo-chondral junctions and Harrison's sulcus, the groove across the lower part of the chest apparently brought about by a folding in this region produced by the "pot belly", which caused the pull of the anterior abdominal muscles to be forward instead of downwards. Pigeon chest did not occur in these mild cases, and when seen in rhachitic children it seemed to be due to abnormal chest conditions rather than to rickets *per se*. The "pot belly" was due to pushing forward of the flabby abdominal muscles by the bowel, which was distended by fermentation of the excess of carbohydrate food so often given to these children. In the second year, a change in the symptoms and signs was noted. Sweating of the head, delay in dentition and late walking and standing were still features. Commonly, the main complaint about the walking child was that it was clumsy and stumbled easily. Dr. Green said that that was due to the laxity of the joint ligaments. He then enumerated the signs in the older child: the fontanelle was still wide; the "pot belly" remained with the chest deformity much as before, but the main changes were now to be seen in the long bones; the epiphyses were splayed out and the joints appeared large; the limbs could be placed in abnormal positions because of the poor muscle tone and the lax joint ligaments. Bowing of the bones and exaggeration of the normal curves might occur, the tibia and fibula and even the femur being affected. If the child crawled much the forearms might become bowed. Some degree of mild anæmia was usually present.

Dr. Green went on to say that these signs were due to the lack of vitamin *D*, which was available to the human in two ways, in foods and in sunlight acting on the skin sterols. In foods there might be a primary lack of vitamin *D*, or the amount might be reduced by staleness or wrong cooking of the food. The lack of the preventive effects of sunlight might be due to non-exposure of the baby from fear of sunburn in summer and from excessive "rugging-up" in the winter. In the city area, it was likely that a loss of the ultra-violet factor in the sunlight occurred, owing to the filtering effect of dust and smoke in the atmosphere. The prevention of rickets, then, lay in the use of good food, with correct balance of protein, carbohydrate and fat. Breast-feeding was preventive if attention was paid to the mother's health, food, exercise and exposure to the sun. Exposure of the baby to sunlight, introduced gradually, was also preventive. In the case of artificially fed babies, the addition of some substance of high vitamin value was essential. For this, the liver oils of cod and halibut and some of the Australian deep-sea fish, irradiated ergosterol or ultra-violet light from a mercury quartz vapour lamp, might be used. Good cod liver oil contained 100 units of vitamin *D* per drachm, and 300 to 400 units daily were adequate for protection. Halibut oil contained up to 500 units per drop, and irradiated ergosterol about 200 units per minim. It had been pointed out that the more concentrated substances might not be adequately absorbed, and in that way some of the poor results of artificial vitamin therapy might be explained. Dr. Green said that his own practice was to use the liver oils with additional concentrate if that was thought necessary. The elimination of rickets from the community called for the education of mothers, nurses and those concerned with food distribution. The knowledge was there, but its application needed much improvement.

Before leaving the subject of rickets, Dr. Green discussed the less obvious problems: first, the tendency of acute illness in the rhachitic child to become chronic, and second, the lack of response to treatment in various ailments unless the underlying rhachitic condition was recognized and adequately treated. Narrowing of the upper jaw had been mentioned; the consequences were important. The high palate caused deflection of the nasal septum, with nasal obstruction; colds were frequent, the antra became infected, and the adenoid pad was chronically inflamed and enlarged. Mouth breathing occurred, and the tonsils, being abnormally exposed, also became enlarged. The narrowing of the jaw further caused crowding of the permanent teeth, which were also of poor quality, as they were developing during the period of active rickets. Hence early caries was almost certain. The rhachitic suffered from severe pertussis and measles with pulmonary complications which failed to clear and led to chronic bronchitis and bronchiectasis. The poor results that followed removal of tonsils and adenoids could often be traced to failure to recognize that the enlargement necessitating their removal was secondary to the malformation of the nose due to rickets. In other diseases such as pyelitis rickets might delay recovery and favour recurrences, whilst in epilepsy, control of the seizures would be achieved more easily if the rhachitic condition often present was treated. While rickets was easily recognized in the rapidly developing infant, it should not be forgotten that the condition would occur in the older child, but that the signs were less evident in these more slowly growing children.

Dr. Green then discussed scurvy. He said that this was the simplest of the deficiency diseases from the clinical standpoint, in that the deficiency of vitamin *C* was followed by the unmistakable train of symptoms and signs of scurvy, while recovery rapidly followed the correct administration of the vitamin. The history given was that a child had been "below par" for some weeks, with fretfulness and pallor, and then the more definite signs appeared. These were due to the hæmorrhagic tendency of the disease. Probably the most common was hæmorrhage from the gums, especially when the baby was teething; the gums were swollen, of velvety appearance and purple in colour, and bled easily, most often about the upper incisors. There might be a history covering several days, of the baby's having cried

if a leg was moved; later the limb was held flexed at the knee and the child screamed if approached, having learnt that any movement of the leg was painful. The pain caused the child not to use the limb; hence the complaint was often that the leg was paralysed. Later, as the hæmorrhage increased, the thigh was swollen and bluish. The hæmorrhage here was subperiosteal and was most common at the lower end of a femur, although other bones such as the radius might be involved. Other hæmorrhages might occur, for example, into the orbit, and red blood cells would commonly be found in the urine, although frank hæmaturia was rare. It was usually stated that bleeding into the skin was uncommon, but in three of the severe cases encountered in 1942 at the Royal Alexandra Hospital for Children petechial spots were present. Scorbutic beading of the ribs occurred, of a different type from that seen in rickets, which was a smooth rounded tumour, due to enlargement of the rib end. In scurvy the edge was sharp, with backward displacement of the cartilage. The raised pulse rate in relation to the normal temperature recalled the similar sign of pink disease. Scurvy occurred at the age of six to twelve months, with the greatest incidence between eight and ten months; the youngest patient recently seen was aged four months, but that was uncommon. Dr. Green then discussed the scurvy admissions to the Royal Alexandra Hospital for Children in the past ten years. The two years with the highest incidence were 1938-1939 and 1942-1943, in which there were 12 and 13 admissions respectively. When these admissions were analysed according to the months of admission, quite different distributions were found. The age grouping was similar for each of the two peak years and conformed to the usual figures for age incidence. Dr. Green said that it was interesting to note that the cases apparently ceased to appear after January, 1943, when there was a public outcry about the number of cases of scurvy. The response was probably shown by the absence of further cases. In the 1938-1939 peak year, no such grouping of cases occurred, and there was no outcry, so that the tendency towards an increase of scurvy in the six years from then on continued. The peak years were probably attributable to the drought conditions of 1938-1939, which affected the orange crop, and to the fact that in the early summer of 1942-1943 the price of oranges was prohibitive.

Dr. Green went on to say that the prevention of scurvy was in the use of an adequate amount of foods containing vitamin *C*. In New South Wales it had become the custom to use orange juice as the standard antiscorbutic, and perhaps this was unwise, as any failure of the orange crop left the public bewildered as to substitutes. It seemed advisable that the various fruits, tomatoes, swede turnips *et cetera* should be used as they came on to the market. Mothers and nurses would thus become accustomed to supplying the infant's needs from the different juices. Ascorbic acid, the synthetic vitamin C_6 when available, was cheap; but again, the use of the naturally occurring vitamin was preferable. The protective dose of vitamin was contained in five to fifteen milligrammes of ascorbic acid (one milligramme equalled twenty international units), and the equivalent amount of good orange juice was two to eight drachms. In treatment, the doses had to be much higher, of the order of 100 milligrammes of ascorbic acid per day, and the equivalent amount of fruit juices would be difficult to give to a sick baby. Therefore a combination of artificial and natural vitamins was used, such as orange or tomato juice, potato or swede turnip with ascorbic acid. Scurvy should be eliminated from the community quite easily; but statistics seemed to show that there was constant need to stress the necessity for added vitamin *C* in infants' diets, otherwise a succession of good and bad periods would occur, the good ones following a "scare" such as had occurred in 1942-1943, with a gradual falling-off in carefulness until another bad period appeared.

DR. H. L. SPEARMAN thought the subject chosen for discussion important. He said that in work at infant out-patient departments one could always pick up a new, undiagnosed case of rickets; the same applied to ariboflavinosis at adult out-patient departments. Dr. Spearman agreed with the speakers that good foodstuffs should be used, rather than vitamin preparations; if a patient was lacking in one part of the vitamin *B* complex, he was likely to be lacking in the whole of it. It was essential to use a natural diet, rather than to prescribe lactoflavine or riboflavine or such things; they were too specific. That was why the deficiency occurred in the first place—the food eaten had, in general, become too purified.

DR. W. L. CALOV said that he thought he should take up the cudgels on behalf of the Section of Medicine, after the mildly disparaging remarks made by Lieutenant-Commander Maddox on the subject chosen for discussion. The choice was not the sole responsibility of the section, though the section heartily approved of it. Lieutenant-Commander Maddox had remarked that frank vitamin deficiency diseases, such as beriberi, scurvy and pellagra, were seldom seen in gross form in Australia, and Dr. Calov thought that quite true. However, minor degrees of vitamin deficiency were very common, as Dr. Spearman had pointed out. Further, as Lieutenant-Commander Maddox had implied, vitamin deficiencies were not necessarily due to lack of vitamins in the diet; the deficiency could be more a question of defective absorption. For example, in ulcerative colitis quite a pronounced vitamin deficiency occurred, and the same was true of sprue. In sprue, the primary fault was a failure of the lacteals of the villi to empty into the lymphatic capillaries; fat was not emptied into the blood stream, and so there was a lack of the fat-soluble vitamins. Later, as a result of inflammation of the whole of the alimentary tract, there was a failure of absorption of other vitamins as well as of various kinds of foodstuffs.

DR. W. C. PETHERBRIDGE referred to the incidence of vitamin deficiency diseases. He said that in treating diseases and in handling normal children, medical men had to survey the diet and see that the subjects received an adequate supply of all vitamins. It was difficult to make sick children take proper food, and as had been pointed out by a previous speaker, the administration of vitamin concentrates did not always give all the vitamins that were required. At the baby health centres it was often found that a child that did badly had a bad parental history; the mother had probably suffered severely from vomiting during pregnancy, or had had to take a restricted diet on account of kidney trouble. The children presented signs of rickets. Dr. Petherbridge also pointed out that another sign of rickets was failure to gain in weight; this might be noticed at the end of the first year, or any time between the ages of six months and two years. Such children had often been well cared for all their lives. If cod liver oil emulsions were "pushed" in such cases, progress was satisfactory.

DR. GRACE CUTHBERT thanked the speakers for their presentation of a difficult subject. She said that she would always welcome any helpful criticism of the work and procedure of baby health centres. It was the object of baby health centres to maintain modern standards and to see that the nurses received educational direction in the latest information concerning the care of children under five. With reference to the increased incidence of scurvy, Dr. Cuthbert said that a senior member of the honorary staff of the Children's Hospital had informed her of the sudden marked increase in the number and the severity of the cases of scurvy being admitted to that hospital. The full facts were ascertained and referred to the State Nutrition Committee. Nurses at the 233 baby health centres in New South Wales were notified of the increasing incidence and a full description of the early symptoms and signs was circulated with the instruction to refer any suspected cases to a medical practitioner and to inform the Department of Public Health. Emphasis was placed on the fruits and vegetables containing the highest percentage of vitamin *C*. The Commonwealth Nutrition Committee was informed and it agreed to publish a double half-column "Food Fact" in the city and country Press. A copy of this "Food Fact" was put up in the waiting room of all baby health centres. The Commonwealth also made available ascorbic acid tablets, 25 milligrammes, for children under two in any district where the natural sources of vitamin *C* had failed. Two cases only had been reported in the last two months, one confirmed radiographically.

DR. E. H. STOKES said that he agreed with Lieutenant-Commander Maddox that gross vitamin deficiencies were not common; but they did occur. It was important that they should be recognized because the results of treatment were usually excellent. Dr. Stokes had found vitamin deficiencies frequently in chronic alcoholics. He thought that he had found a case of the rare condition referred to by Lieutenant-Commander Maddox, beriberi heart. The patient was a man who consumed sixteen pints of beer a day; he had tachycardia, œdema, polyneuritis *et cetera*. The man did well when he was taken off the beer and put on vitamin *B* therapy. Dr. Stokes said that other manifestations of alcoholism were also well handled by the exhibition of vitamin *B*. Scurvy also occurred in alcoholic people, because alcoholics did not as a rule consume enough food-containing vitamin *C*.

DR. E. H. M. STEPHEN said that he wished to draw attention to an old-fashioned remedy for scurvy—the use of potato cream. The potato, humble as it was, was a very

obliging vegetable, much more easy to grow than the much vaunted tomato, and obtainable in all places at all times. Dr. Stephen said that the method of preparation was to cook the potato in its jacket, which prevented deterioration of vitamin *C*. On removal of the skin, the substance beneath it was mixed with milk and butter to a cream. Two ounces of this at least were given daily. It was palatable and devoid of that acidity which some children resented in tomatoes and fruit.

Surgeon Lieutenant-Commander Maddox said that he felt that the subject could have been much better dealt with if it had been spread over two or three meetings and discussed by nutrition experts including dietitians. Lieutenant-Commander Maddox still felt that larger daily amounts than 80 units of vitamin *D* were necessary as a prophylactic against rickets, because the question of absorption, as Dr. Green had said, was so uncertain that it was necessary to provide a fairly safe margin. One thousand units had been recommended by American authorities. Dr. Petherbridge had referred to pre-natal influences causing rickets; it was interesting to find that toxæmia of pregnancy itself had been held by some authorities to be a vitamin deficiency disease. However, others had denied the hypothesis, which appeared to be losing ground. Rickets did seem to occur more often in children of mothers who had had a disturbed pregnancy. Lieutenant-Commander Maddox then said that there were other sources of vitamin *C* than the commonly used ones; for example, tomato juice, swede turnip juice and the juice of papaws were very good and could be used canned. Black currant juice also was an excellent source of the same vitamin. Dr. Stokes had referred to the value of vitamin *B* therapy in polyneuritis due to alcoholism; Lieutenant-Commander Maddox said that the whole question was becoming rather involved. It was now less certain that a vitamin deficiency was the whole story in alcoholic polyneuritis. Some authorities had found that large doses of thiamin had not reduced the period of convalescence so much as would have been expected, and in Boston it had been found that alcoholics had to remain in hospital just as long at the present time as before thiamin treatment was introduced. Some other factor seemed to be present. The theory that œdema was due to vitamin *B* deficiency had also come in for criticism at the present time, and hypoproteinæmia had been brought forward as the explanation. Lieutenant-Commander Maddox had seen as patients two naval officers, who had been in the bush around the Milne Bay area for as much as three months at a time; they had taken some time to respond to vitamin *B* therapy. But when a diet rich in protein was given, the second of the two patients seemed to recover much more quickly. Lieutenant-Commander Maddox said that Dr. Stephen's remarks about the potato were very apposite; the potato was the main source of vitamin *C* for the man in the outback and for his family. Actually, potatoes contributed quite a considerable proportion of vitamin *C* to the diet. On destroyers and other small ships, where it was not possible to store for very long large quantities of food containing vitamin *C*, the cooks could produce quite good salads from potato peelings. By the time the potatoes were scrubbed and the peelings were redressed, they were scarcely recognizable for what they were.

Dr. Green, in reply to Dr. Spearman, who had referred to the provision of an adequate diet, said that was a matter of educating the mothers and nurses. In that regard, Dr. Green took the opportunity of paying a tribute to the work done by the baby health centres of New South Wales. The fact that Dr. Cuthbert was able to mobilize such a body of opinion when an outbreak of scurvy occurred emphasized the good work that was being done. Dr. Green then referred to Dr. Petherbridge's remarks about the child who failed to gain in weight at about the end of the first year. Dr. Green said that such a phenomenon in nearly all cases indicated that some minor vitamin deficiency was present, usually vitamin *D*. Anæmia occurred easily in these young children. In conclusion, Dr. Green said that only lack of time had caused him to fail to mention the potato as a potent antiscorbutic.

DR. K. S. M. BROWN, from the chair, said that he thought that the subject chosen by the Section of Medicine for discussion that night was very appropriate. It was a good thing for the medical profession to be reminded once again of the deficiency diseases. Dr. Brown had noticed a serious deficiency in his own medical armamentarium, and that was a lack of knowledge of the basic principles of nutrition and dietetics. He thought that that knowledge was probably lacking among a number of his colleagues in practice. No doubt the younger men received very sound teaching in biological chemistry and would be taught how to advise their patients in such matters as diet. The subject would become more important in the future. The general public had much more knowledge at the present time, and that was due to the information given by the baby health centres and to the broadcast talks under the title "The Kitchen Front", which were given every morning. The public expected to be guided in the principles of nutrition with a view to acquiring "positive health". In conclusion, Dr. Brown said that the members of the medical profession needed to be reminded again of the necessity for more knowledge of these deficiency diseases, and on behalf of those present he thanked speakers and those who had joined in the discussion.

Correspondence.

AN UNUSUAL EYE CONDITION.

SIR: The following rather unique case presents several points of topical and general interest.

Mrs. O.H., aged 22, was surfing at Merewether Beach on October 22 last, when she was stung fairly extensively by a Physalia (blue-bottle, Portuguese man-o'-war). Her chief complaint was about her right eye, on which account she attended the hospital casualty department, where argyrol 15% drops were prescribed.

I saw the patient first on the following day, when she was complaining of considerable pain still. Examined with a loupe and focal illumination, the cornea appeared clear, there was no staining with fluorescein, the pupillary reactions were normal, and there was no marked photophobia. The only positive objective finding was a very slight ciliary injection, in view of which, and in consideration of a complete ignorance of any such case, atropine 1% was instilled and the eye kept covered.

Two days later (the third day) the eye was still very sore. There was now no ciliary injection, again no staining, and the whole appearance was completely quiet. But with the slit-lamp there was seen to be a slight crenation and œdema of the whole of the corneal epithelium. *Unguentum Atropini* 1% was ordered twice a day.

On the fifth day the eye still looked quiet, but the slit-lamp revealed widespread degenerative changes in the corneal epithelium, now with many minute staining points. At this stage the appearance was very akin to the early vesicular degeneration in a *herpes simplex* ulceration, and also very like a fine superficial punctate keratitis. The corneal nerves were remarkably prominent, and in view of the continued pain, *Unguentum Metaphen cum Butyn* 2% was prescribed.

By the seventh day the epithelium had repaired entirely and there was no staining, but the surface was studded with fine glistening bodies, possibly crystalline, but too small to describe even when seen under the binocular microscope. The pain was very much less and the patient was advised to suspend the butyn ointment unless the pain returned. The complaint was now that the eye felt "gritty".

After two weeks the appearance was still much the same, though the number of surface particles was less.

At the end of the third week the eye still had the gritty feeling and there was some pain. There was now a roughening and irregularity of the corneal stroma in the lower segment, without ulceration. This was considered to be a trophic change, again resembling some of the lesions seen in a *herpes corneæ*.

By the end of five weeks the eye was entirely comfortable and the only residuum was a slight prominence of the corneal nerves in the lower part. The atropine was suspended and the eye uncovered. There has been no further trouble and the acuity is $^6/_6$.

Conclusions.

1. The long delay in the appearance of demonstrable effects is remarkable. Even after three weeks an active degenerative process was in evidence.

2. The duration of the case is some five weeks, a period more to be considered in terms of an infective than a chemico-toxic agent.

3. Without a knowledge of the history, the clinical picture would easily have been ascribed to a neurotropic virus, and no doubt the toxins liberated by such infections are similar in effect to the neurotoxin of the blue-bottle.

4. Pain was a prominent feature and seems to have been due to a local action, since it was relieved by local anæsthesia with butyn. Contrast *herpes zoster*.

5. Except for the initial very slight ciliary injection, there was a complete absence of vascularization. Is this the reason for the prolonged duration of the case?

6. Recovery was complete without any specific treatment.

Finally, I should like to thank Dr. Arthur D'Ombrain, with whom I was in practice at the time, for his help and advice, and for permission to report the case.

Yours, etc.,

JOHN HERCUS.

Bank Chambers,
17, Bolton Street,
Newcastle,
New South Wales.

December 23, 1943.

THE SERVICES AND CIVIL PRACTITIONERS.

SIR: Whilst in agreement with some of the points raised by your correspondent, "R.M.O., First Australian Imperial Force", in his letter in your issue of January 1, I feel some of the questions raised by him call for a reply.

R.M.O. states: "It has been said (and some think with justice) that no young medical officer who is fit should have a base job in the air force until he has had a period of active service with the army or navy."

A statement such as this shows a lack of appreciation of the organization and the work of the Royal Australian Air Force Medical Services. For success to be achieved in modern warfare the most complete mutual cooperation and support must exist between the three services right up to the front line, and this has always been so as far as our medical services are concerned.

It is not permitted to quote the actual numbers of Royal Australian Air Force medical officers who are in forward areas, but a percentage analysis shows:

1. That 36% of all Royal Australian Air Force medical officers are at present on duty in forward operational areas.

2. Seven *per centum* are on overseas service with squadrons and other units in almost every theatre of war.

The policy in the Royal Australian Air Force is to give every fit medical officer the opportunity of service in forward areas, and by rotation of duties between these officers and those needed for duty at hospitals and units on the mainland to spread the opportunities for active service and for clinical experience as widely as possible.

Already more than 50% of all Royal Australian Air Force medical officers have served or are serving in forward areas, and with the continued expansion of the service and the extension of the area of hostilities, there will soon be very few fit medical officers who have not seen active service in operational areas.

It has to be borne in mind that in order to provide reliefs for medical personnel in forward areas there must, of necessity, be sufficient personnel under training on the mainland to relieve them.

In view of the very special medical problems associated with operational flying, there must always be a sufficient number of medical officers on the mainland undergoing training and gaining experience on flying training units before proceeding to operational squadrons.

The question of organized post-graduate work for service medical officers has been given earnest consideration by the services' medical directors and recommendations for a comprehensive post-graduate scheme have been submitted to the appropriate authorities.

Yours, etc.,

VICTOR HURLEY,
Air Vice-Marshal, Director-General of Medical Services, Royal Australian Air Force.

January 6, 1944.

Post-Graduate Work.

WEEK-END COURSE AT WOLLONGONG.

THE New South Wales Post-Graduate Committee in Medicine announces that, in conjunction with the Clinical Society, South Eastern Medical Association, it will hold a week-end course at Wollongong on Saturday, February 5, and Sunday, February 6, 1944. The programme is as follows:

Saturday, February 5.

At the Wollongong Hospital.

2 p.m.: Registration.
2.30 p.m.: "Volvulus Chronic Intussusceptious Intestinal Obstructions", Dr. Robert Malcolm.
4 p.m.: "Heart Disease", Dr. Bruce Shallard.

Sunday, February 6.

At the Wollongong Hospital.

10 a.m.: "Chemotherapy with Reference to the Sulphonamide Drugs", Dr. Bruce Shallard.
11.30 a.m.: "Strangulation of Abdominal Organs", Dr. Robert Malcolm.
2 p.m.: "Modern Hospital Service", Dr. A. Lilley.
3 p.m.: "Treatment of Burns", Colonel K. Starr.

The fee for the course will be £1 1s., except for members of the defence forces, who may attend the course without fee. Those intending to be present at the course are requested to notify Dr. R. A. Green, Honorary Secretary, Clinical Society, South Eastern Medical Association, Port Kembla, as soon as possible.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes et cetera have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 2 and 7, of January 6 and 13, 1944.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointment.—John Mansfield Newton is appointed Temporary Surgeon Lieutenant (D), dated 5th December, 1943.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Grant of Acting Higher Rank.—Surgeon Lieutenant Richard Fitzwalter Read is granted temporarily the acting rank of Surgeon Lieutenant-Commander, dated 24th February, 1943.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The following officers are transferred from the Medical Branch Reserve to the Active List for full-time duty with effect from the dates indicated: (Flight Lieutenants) C. B. Colvin (267224) (1st November, 1943), A. G. Gibson (256822), J. K. L. Marks (7392), A. G. Nicholson (257529) (14th November, 1943).

Flight Lieutenant F. E. Browne (256800) is transferred from the Medical Branch Reserve to the Active List for part-time duty with effect from 30th September, 1942.

The previous notification concerning Flight Lieutenant F. E. Browne (256800), which appeared in *Commonwealth of Australia Gazette* No. 325, dated 17th December, 1942, is cancelled.

The following Flight Lieutenants are granted the acting rank of Squadron Leader whilst occupying Squadron Leader posts with effect from the dates indicated: J. F. Ziegler (257480), 1st October, 1943, S. MacK. Morson (262063), 1st December, 1943.

Flight Lieutenant J. D. Whiteside (256812) is transferred from the Medical Branch Reserve to the Active List with effect from 1st October, 1942.

The probationary appointments of the following Flight Lieutenants are confirmed: A. P. Roberts (266395), C. K. Hemmingway (266936), C. S. Harper (297386), T. E. H. Spark (267437), W. J. Stevenson (257483), G. J. B. Phillips (267374), T. J. Lowe (266786), J. A. Edye (266715), J. T. Cullen (267165), I. M. Lilley (257076), N. G. Dobell-Brown (267113), I. C. Morrison (266409), H. S. Moore (266931), C. R. Watson (266788), A. I. Lane (266930), J. B. Maloney (267436), A. K. Smith (267110), F. J. McCoy (253474), H. S. Moroney (254365), A. L. Hare (254419).

Reserve: Medical Branch.

Flight Lieutenant T. P. Mahon (261455) is transferred from the Active List as from 6th November, 1943.—(Ex. Min. No. 5—Approved 12th January, 1944.)

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the

dates indicated: Leonard George Knott, M.B., B.S. (277475) 28th September, 1943), Russell Roxburgh, M.B., B.S. (267591) (19th October, 1943).

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from 10th November, 1943: Christopher Lancaster, M.B., B.S. (257597), Charles Ronald Wherrett, M.B., B.S. (267599), George Whyte, M.B., B.S. (267598).

The following officers are transferred from the Active List with effect from 6th December, 1943: Squadron Leader I. R. Horn (271202), Flight Lieutenant G. L. Young (261771).—(Ex. Min. No. 16—Approved 12th January, 1944.)

CASUALTIES.

ACCORDING to the casualty list received on January 20, 1944, Captain R. E. R. Gillespie, A.A.M.C., who was placed on the "seriously ill" list, is now reported to have been removed from it.

Obituary.

LESLIE DAVIES.

WE regret to announce the death of Dr. Leslie Davies, which occurred on December 8, 1943, at Melbourne.

SYDNEY MICHAEL O'RIORDAN.

WE regret to announce the death of Dr. Sydney Michael O'Riordan, which occurred on January 19, 1944, at Sydney.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Gillogley, Jack Francis, M.B., B.S., 1941 (Univ. Sydney), 60, King Street, Ashbury.

Hill, Kenneth Hamilton, M.B., B.S., 1939 (Univ. Sydney), 2, Victoria Street, Strathfield.

Kiely, Roger John, M.B., 1941 (Univ. Sydney), 26, Crescent Street, Haberfield.

Byrne, Ian Donald, M.B., B.S., 1943 (Univ. Sydney), Balmain and District Hospital, Booth Street, Balmain.

Vickers, Thomas Harold, M.B., 1942 (Univ. Sydney), Residents' Quarters, Sydney Hospital, Sydney.

Corrigendum.

AN ERROR IN SULPHAGUANIDINE ADVERTISEMENT.

Leyshon Publicity Services desire to correct a mistake made by them in the preparation of an advertisement on behalf of Nicholas Proprietary Limited in THE MEDICAL JOURNAL OF AUSTRALIA ADVERTISER of January 15, 1944.

The heading "Vitamin Therapy" in the sulphaguanidine advertisement was inserted in error.

Medical Appointments.

His Excellency the Governor in Council has been pleased to appoint the undermentioned persons to be resident medical officers at the Royal Adelaide Hospital: William Ross Adey, Gilbert William Elliot Aitken, Colin Graham Alderman, Max Kimberley Anderson, Arthur William Burnell, Robin Archibald Burston, David Edward Craven, David Llywelyn Davies, Murray William Elliott, Ronald Munro Ford, Marcus Gordon Jansen, Herman Frederick Kuhlmann, Peter William Leslie, David Archibald Stevenson Morgan, Ian Gordon Pavy, James Alexander Bryan Rolland, Robert Alfred Russell, Kenneth Villiers Sanderson, John Hamilton Stace, Gayfield Collins Thornton.

Books Received.

"Forsdike's Textbook of Gynæcology", revised, enlarged and in part rewritten by J. H. Peel, M.A., B.M., B.Ch. (Oxon), F.R.C.S., M.R.C.O.G.; 1943. London: William Heinemann (Medical Books) Limited. 8½" × 5½", pp. 452, with many illustrations. Price: 21s.

"Manual of Psychological Medicine: For Practitioners and Students", by A. F. Tredgold, M.D., F.R.C.P., F.R.S.E.; 1943. London: Baillière, Tindall and Cox. 8½" × 5½", pp. 307. Price: 18s.

Diary for the Month.

JAN. 31.—Federal Council: Meeting at Melbourne.
FEB. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee Special Groups Committee.
FEB. 2.—Western Australian Branch, B.M.A.: Council Meeting.
FEB. 2.—Victorian Branch, B.M.A.: Branch Meeting.
FEB. 4.—Queensland Branch, B.M.A.: Branch Meeting.
FEB. 4.—Victorian Branch, B.M.A.: Legislative Subcommittee.
FEB. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 11.—Queensland Branch, B.M.A.: Council Meeting.
FEB. 11.—Victorian Branch, B.M.A.: Ethics Subcommittee.
FEB. 14.—Victorian Branch, B.M.A.: Hospital Subcommittee.
FEB. 14.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be Stated.

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